PB-216 113

PROCEEDINGS OF THE NATIONAL CONFERENCE ON SALMONELLOSIS HELD AT THE COMMUNICABLE DISEASE CENTER IN ATLANTA, GEORGIA ON MARCH 11 THROUGH MARCH 13, 1964

James L. Goddard

Communicable Disease Center
Atlanta, Georgia

March 1965

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PROCEEDINGS

NATIONAL CONFERENCE ON SALMONELLOSIS

MARCH 11-13, 1964

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Public Health Service Publication No. 1262
March 1965

U.S. GOVERNMENT PRINTING OFFICE
WASHINGTON : 1965

FOREWORD

In light of the extent and importance of the salmonellosis problem in the United States, the Communicable Disease Center sponsored a National Conference on Salmonellosis that was held in Atlanta, Georgia, March 11 through March 13, 1964. Over 200 representatives from State and Federal agencies, industry, and universities attended. The multiple interrelated facets of the problems associated with salmonellosis were presented in formal papers and discussed in informal sessions.

The interchange of ideas that transpired was instrumental in opening channels of communication, necessary to the development of a successful salmonella control program.

This monograph is the record of the conference, and includes the formal papers presented and the pertinent data reported during the several discussion sessions.

James L Goddard

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Welcome

David E. Price, M.D.[1]

It is a pleasure to welcome each of you to the Communicable Disease Center for this National Conference on Salmonellosis. Though you represent a variety of skills and interests with diverse backgrounds and training, there is one unifying factor that has brought you here together—widespread concern about salmonellosis. Not only does interest in this disease cut across professional lines, but it also crosses State and national boundaries. Among the approximate 225 individuals attending this conference, there are 69 people representing State departments of public health, agriculture, and food and drug regulation; 46 are from the U. S. Public Health Service; 31 from other Federal agencies; 32 from industry; 22 from universities; 6 from city health departments; and 6 from hospitals. Eight distinguished experts from other countries have been invited to give us the benefit of their knowledge of salmonellosis.

The true incidence of salmonellosis in the United States is unknown. Surveillance data reported from the States, however, indicate that the number of salmonella isolations from humans is increasing. There is good reason to believe that for every case of salmonellosis reported, there are 5 or 10 or 20 undiagnosed but related cases. Salmonellosis is truly a major public health problem of concern to people in every walk of life.

There is no easy approach to solving the complex problems leading to the control of this disease. In investigating cases of salmonellosis, it is not enough to just identify the cases and any carriers and report the findings to the local health officials. This initial effort should serve as an impetus to trace the route of infection back to its source, describe the ecology of salmonellosis, and help prevent future cases. The pathways are not necessarily obvious and, in fact, may be so circuitous that tracing back becomes impossible. The attempt must be made, however; and, when the route or routes have been identified, another brick can be placed in the fortress of prevention.

Each of you must assume a role of leadership in attacking this problem. Each of you must stand up to your responsibilities and, within your own sphere of activity, do all you can to assist in externalizing our knowledge of this disease. We hope that, when you return home, you will discuss the data presented at this meeting with your colleagues so that all with a role in investigations and control of salmonellosis will have an opportunity to benefit from these discussions. A monograph of this conference will be prepared and distributed to all in attendance.

The Public Health Service recognizes its responsibilities in the study and control of salmonellosis. Both its Communicable Disease Center and its Division of Environmental Engineering and Food Protection are vitally concerned. The Food and Drug Administration, our sister agency in the Department of Health, Education, and Welfare, is also importantly involved. Other Federal departments share our keen interest, and we are particularly pleased to be joined here by representatives of the Departments of Agriculture and Commerce. All of these agencies are willing to cooperate with State or local officials in any activity that is related to the control of salmonellosis.

The Communicable Disease Center is a logical leader in spearheading surveillance and investigations. Its forces consist of epidemiologists, nurses, and veterinarians, to assist in field studies. It has laboratories to assist in improving diagnostic laboratory procedures; to provide reference and typing services, consultation, assistance, and training; and to supply biological reagents. It has training facilities to instruct in all aspects of public health by means of programs either here at the CDC or in the States, and audiovisual facilities are available to prepare a wide variety of training aids. Its Technology Branch is staffed to evaluate and instruct on practical and acceptable control measures. We have worked with many of you during the past years and continue to offer our assistance to each of you in the future. This must be a team effort with everyone concerned participating to the best of his ability.

Thus, we welcome you to the Communicable Disease Center and hope that while you are here, any strangers will become acquainted with the Center and all of its operations. A tour of the Center or a meeting with a certain branch or individual can be arranged at your convenience. Each of your salmonella problems are different; the solutions will vary; but the end result of adequate control is a universal objective. If the presentations and discussions during the next 3 days are of assistance in helping you approach the resolution of your problems, then this conference will have been successful.

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[1] Deputy Surgeon General, Public Health Service, U.S. Department of Health, Education, and Welfare, Washington, D.C.

Purposes of Conference

James L. Goddard, M.D.[1]

It is paradoxical that the incidence of salmonellosis in the United States continues to rise in spite of the continued long-term interest in the problem. This interest is evident in the field and research studies which have been reported during past years and the continued improvements which have been designed to reduce the hazards of food-borne disease in general and salmonellosis in particular. Is this an indication that we do not understand the ecology of this disease? Are effective methods of control still lacking, or have they been developed but not adopted by those in a position to institute control procedures?

It is our hope that by bringing together representatives from industry; local, State, and Federal agencies; and universities to discuss various aspects of salmonellosis that the voids in our knowledge can be separated from the known facts. Accordingly, investigative teams can continue to consider methods and means of exploration in unknown areas, while current knowledge can be utilized in the development of effective and practical control procedures.

We must remember that enough data is already available concerning some aspects of contamination of foodstuffs so that several countries and at least one State in the United States have written or are writing regulations prohibiting the use of certain products unless they are certified salmonella-free. I hope that those of you acquainted with these activities will report on them at some time during this conference.

This conference has been designed to promote the maximum transmission of information among all participants. The program has been arranged in an attempt to cover the most important aspects of salmonellosis as a public health problem. The speakers have been selected because of their known interest and active participation in studies related to some aspect of salmonellosis.

Five to 10-minute discussion periods have been allotted following each presentation. Additionally, three 1-hour panel discussions have been planned, one on each of the conference days, so that a panel of experts can answer questions raised from the floor. I am sure additional discussion will follow at coffee breaks, and in the evenings; and, hopefully, lines of communication will have been established during the 3 days, which will continue after the conference.

During the first day, we shall delineate the problem of salmonellosis not only in the United States, but in Canada and the United Kingdom, where problems are similar to ours. The problems of animal disease and human disease are closely interwoven and will be discussed at the same time. The afternoon program will cover the various population groups at risk and give us an insight into some of the problems of identifying outbreaks. This will be followed by the first of the panel discussions.

On the second day, a series of papers will explore the variety of sources of salmonella which should indicate the universality of the organism. A second panel will then assemble to discuss additional questions. The afternoon will be devoted to discussing the recent, and in some respects, continuing interstate, hospital epidemic of Salmonella derby gastroenteritis. This outbreak actually can serve as a focal point for many of the presentations at this conference.

A major goal is to explore the area of control. This will be discussed on Friday morning by representatives from the Federal government and from industry. A third panel discussion will follow with the panel members drawn from both groups. The speakers from industry have been asked to be representative of the entire industry as distinct from their own company.

We have arranged a postconference seminar on laboratory methodology for Friday afternoon, which will deal with field and laboratory procedures for collection, isolation, and identification of salmonella from feeds, food, and environment. Following a panel discussion, there will be a laboratory demonstration to exemplify the methods described.

The problems are complex; the solutions involve more than the efforts of one person or company or agency. They involve the cooperative efforts of the multiple disciplines concerned. There must be a spirit of cooperation if this major public health problem is to be approached with dignity and dispatch. Control is not necessarily based upon new or undiscovered concepts, but to a large extent can be accomplished with recognition of facts and application of principles that are based upon our current knowledge. The benefits of this conference must be yours and can only result from cooperative work among all peoples involved. If this can be accomplished with this conference serving as a catalyst, then we have been successful in our purpose.

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[1] Assistant Surgeon General, Public Health Service, U.S. Department of Health, Education, and Welfare, and Chief, Communicable Disease Center, Atlanta, Georgia.

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DELINEATION OF THE PROBLEM

MODERATOR : ***Dr. James L. Goddard***

Salmonellosis - An Expanding Problem

Philip R. Edwards, Ph.D.[1]

In opening a discussion on the subject assigned, no remark could be more pertinent than one contained in a recent issue of a commercial publication, to wit: "Salmonella bacteria are everywhere." [2] Such a statement regarding the ubiquity of salmonellae, while exaggerated, succinctly states the problem with which we are faced today and which, at least in part, provided motivation for the present conference. It well might be said that those of us who are concerned with the problem of salmonellosis, presently suffer from an embarrassment of riches. This situation stems largely from advances in two areas in the study of salmonellae: first, marked improvement in methods of isolation of the organisms; and second, evolution of methods whereby etiologic entities can be distinguished through division of the organisms into serotypes and subdivision into phage types.

The development of numerous modifications of enrichment and plating media, which permitted the isolation of salmonellae from a much larger number of specimens and from more diverse sources than previously was possible, resulted from the observations of many investigators. While the names of MacConkey, Wilson and Blair, Kristensen, Kauffmann, Leifson, and others are closely connected with the almost innumerable modifications of media presently used, it must be remembered that these media developed gradually and that their origins go far back into bacteriological literature. Likewise, the investigations of White and of Kauffmann, which are the basis of our present methods of distinguishing salmonella serotypes, are the culmination of the work of many persons, extending to the very early years of the 20th century. Thus, the aforementioned "embarrassment of riches" resulting from numerous isolations of salmonellae from a broad spectrum of sources in which their presence previously was unsuspected, as well as the increasing number of etiologic entities which can be distinguished, has not burst suddenly upon us but has developed gradually over the years. In other words, the expanding problem of salmonellosis is by no means a new one, and our present concern with it is due to increased awareness of its existence and realization that control measures should be instituted.

Increased attention to the problem of salmonellosis probably stems from two sources. First, the most important host-adapted salmonelloses of man and fowls are diminishing rapidly in incidence; and, since we no longer are so preoccupied with their control, our attention has turned to related infections. Table 1 shows the reported incidence of typhoid fever and of other salmonelloses of man in the United States from 1946 to 1963. The incidence of typhoid fever has fallen steadily; whereas reports of other salmonelloses have increased more than twentyfold. The decrease in incidence of typhoid fever is an excellent example of the control which can be achieved in the case of a strictly host-adapted type through thorough study of its ecology and methods of transmission and through exercise of all possible measures to restrict its spread. Pullorum disease well could be cited as a similar example among the salmonelloses of animals. In contrast, the non-host-adapted types now

Table 1.–*Typhoid fever and other salmonellosis (reported cases by year, United States)*

Year	Typhoid fever	Other salmonelloses
1946	3,268	723
1947	3,075	951
1948	2,840	882
1949	2,795	1,243
1950	2,484	1,233
1951	2,128	1,733
1952	2,341	2,596
1953	2,252	3,946
1954	2,169	5,375
1955	1,704	5,447
1956	1,700	6,704
1957	1,231	6,693
1958	1,043	6,663
1959	859	6,606
1960	816	6,929
1961	814	8,542
1962	608	9,680
1963	525	18,696

---

[1] Chief, Bacteriology Section, Laboratory Branch, Communicable Disease Center, Public Health Service, U.S. Department of Health, Education, and Welfare, Atlanta, Georgia.

[2] From Frontiers in Nutrition, Supplement, No. 152, p. 579, December 1963. The Dawes Laboratories, Chicago.

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are being recognized more frequently in man and animals than previously. It is not meant to infer that the rather dramatic increase in the number of salmonella infections recognized is due wholly to increased incidence. Increased awareness of, and interest in, the infections and use of improved methods of diagnosis undoubtedly are partially responsible, yet reporting of the actual number of cases still is woefully incomplete. The southeastern United States presents an excellent example of the incomplete reporting that exists in some areas. In 3 States within this area, reported cases per capita for December 1963 were 8 times as great, and, in 2 States, 21 times as great, as those in 4 other States of the section. Similar or greater differences involving the same States occur repeatedly. Such differences in the actual occurrence of salmonellosis obviously do not exist in neighboring States. Only differences in the concern of the medical and public health professions with the problem are responsible.

Table 2 shows the reported incidence of general outbreaks of salmonellosis per year per 100,000 population in the United States and in England and Wales. The figures indicate how ridiculously low the reporting in the United States has been in the past. The ratio of 1 to 28 for the years 1953 through 1955 is a reflection of the better reporting and more intensive investigation of cases in Britain, since the outbreaks recorded are those which involved more than one family. From 1953 through 1962, there was no significant change in outbreaks reported in the United States; but in England and Wales, although the system of reporting apparently remained unchanged, there was a significant reduction in reported outbreaks. The doubling of the reported outbreaks in the United States in 1963 is undoubtedly the result of better reporting resulting from the salmonella surveillance program now in effect in all the States. It is rather remarkable that, at the time the ratio of reported outbreaks of salmonellosis was 1:28, that of staphylococcic food poisoning was only 1:2.

Table 2.–*Salmonellosis general outbreaks per year per 100 thousand population*

Years	United States	England and Wales	Ratio U.S.:England and Wales
1953-1955	.014	.390	1:28
1959-1961	.010	.183	1:18
1962	.013	.117	1:9
1963	.026	(1)	(1)

[1] Not available

The second source of concern regarding salmonellosis is that, while it is extremely difficult to document, it is felt by most persons working in the field that there has been an actual increase in salmonellosis in both man and animals. It is thought that the suspected real increase is due in large part to the changing food habits of the human and animal populations and to improved systems of distribution which lead to a wider dissemination of individual products which act as vehicles. Technological progress in the preparation and distribution of foods and feeds has been rapid; and, in many instances, our methods of control and measures for their enforcement have not kept pace. In some areas, there has been a reluctance to establish official microbiological standards even though they would have been welcomed by industry.

The rapidly increasing numbers of recognized salmonella serotypes have caused some individuals to become discouraged regarding control of salmonellosis. The number of recognized types has increased from 44 in 1934 to well over 800 at the present time, and hitherto unrecognized serotypes have been appearing at the rate of 50 to 80 each year. This situation is the result of much more extensive study of the ecology of the genus and also of intensive bacteriological exploration of previously unexplored geographical areas which invariably leads to a rash of new serotypes. While the recognition of many new antigenic components and antigenic combinations complicates the work of laboratories engaged in the exact characterization of serotypes, the situation regarding the average diagnostic laboratory remains unchanged. As shown in Table 3, the vast majority of salmonellae encountered by a reference laboratory are members of a small number of O antigen groups and a relatively small number of serotypes. This table, which is based on cultures received at the Communicable Disease Center over a period of 10 years, illustrates that serotypes which occurred more than 30 times made up 96.7 percent of the material and were divisible into only 55 serotypes. Among cultures from man, 39 serotypes accounted for approximately 95 percent of the total. A much larger number of rarely occurring serotypes accounted for only a very small proportion of the cultures. It should be noted that most of the latter belonged to the same O antigen groups represented by the frequently occurring types. The situation further is alleviated by the fact that in recent years simple biochemical tests have been devised which permit rapid recognition of members of the genus even in the small laboratory. In the United States, availability of reference services at State level has reached the point that no difficulty should be experienced in diagnosis through application of readily available materials and methods. In recent years, much has been said concerning transduction, lysogenic conversion, and conjugation in the salmonellae with consequent changes in biochemical and antigenic properties. In some quarters, questions regarding the effect of these processes on diagnostic bacteriology have been voiced. There is no reason to believe that these are new phenomena or that the microrganisms suddenly

Table 3.—*Distribution of O groups and serotypes in diagnostic material, July 15, 1947–July 15, 1957*

Number of cultures per type	O groups	Number of serotypes	Number of cultures	Percent of total
All sources:				
Greater than 50	B through E_4 + G	41	22,132	94.5
Greater than 30 but less than 50	A through H + K + N	14	510	2.2
Less than 30		146	772	3.3
Total		201	23,414	100.0
Man:				
Greater than 50	B through G	31	11,307	92.2
Greater than 30 but less than 50	A through G + K	8	313	2.6
Less than 30		152	647	5.2
Total		191	12,267	100.0

have become more sophisticated than in the past. Diagnostic difficulties should not be expected to increase due to advances in our knowledge, but rather to decrease.

It is not meant to infer that, from an epidemiological standpoint, all diagnostic difficulties have been overcome. Progress in the application of bacteriophage typing for subdivision of serotypes into phage types, which can be more closely associated with etiologic entities, has been slow. While phage typing has been applied to *Salmonella typhi* and *S. paratyphi B* for a number of years, application of the procedure to other serotypes is relatively new, and practical use of the method has been extended only to *S. typhi-murium*. Even in this instance, the work is hampered by lack of readily available phages of desired quality and range and by lack of trained personnel to accomplish the task. Approximately 600 cultures of *S. typhi-murium* are reported in the United States each month. To phage type all of them in one or a few laboratories would be a sizable task, and the problem of propagation and distribution of suitable phages as yet has not been solved. Future extension of the system to other frequently occurring serotypes should be attempted. It should be said, however, that phage typing of salmonellae, while contributing to the solution of individual epidemics, will not be of great aid in the practical control of salmonellosis until obvious sources of dissemination are attacked and the infections brought to a much lower level of prevalence than now exists.

The ever broadening search for the presence of salmonellae and the increasingly intensive methods employed in their detection have added to our knowledge of the ecology of the group and led to their discovery in previously unsuspected environments. Their presence in the intestinal tract of a significant number of reptiles — including turtles, snakes, and lizards — and their persistence in some of these animals for several years were unexpected findings. The repeated isolation of salmonellae from foods of vegetable or marine origin indicates the many opportunities for contamination that occur in the processing of originally uncontaminated materials and the multiple pathways by which the organisms may gain entrance to foods. Coconut, soya milk, and dried yeast are examples of such products. The isolation of salmonellae from many other sources could be mentioned; but the fact remains that, although almost 80 years have elapsed since the recognition of food-borne salmonellosis, we still have much to learn regarding the ecology of the organisms. Nevertheless, there is at hand sufficient knowledge of their habitat and methods of their dissemination to do much more in the control of the infections than presently is being done.

A voluminous literature has accumulated regarding the presence of salmonellae in foods, particularly foods of animal origin. It long has been known that salmonellae could be isolated from the mesenteric lymph nodes of normal swine and that in this location they occurred in amazing numbers and variety. The role of the holding lot and the abattoir in the transmission of the organisms in swine and in subsequent contamination of carcasses has been investigated intensively. Further, it has been demonstrated repeatedly that contamination of carcasses in the abattoir is reflected by salmonellosis in local populations and in carrier rates among abattoir employees. The widespread outbreaks of salmonellosis which have occurred in Sweden and the Netherlands within the past decade were traced to beef carcasses which were contaminated in abattoirs and shipped without adequate refrigeration. It has been demonstrated that it is possible to reduce the contamination of carcasses by thorough cleaning of abattoirs and treatment of carcasses. It is well known that carcasses and edible viscera of poultry often are contaminated with salmonellae; but, in poultry plants, contamination seems to be intermittent and to depend upon the presence of salmonellae in birds being processed. This situation contrasts with the rather constant presence of the organisms in abattoirs in which swine are slaughtered and is probably due to differences in processing and the equipment involved.

The presence of salmonellae in eggs and egg products long has been a source of concern, and egg products often have been the source of contamination of prepared foods and food mixes which have given rise to salmonellosis. One has only to review the recent experience in Canada where *S. thompson* has been found so predominantly in food mixes that contained egg, some of which are known to have given rise to overt salmonellosis, and where this organism has replaced *S. typhi-murium* as the serotype found most frequently in salmonellosis. The frequent occurrence of salmonellae in egg products has given rise to legislation in attempts to ensure freedom from contamination. The postwar Swedish embargo on egg products in which salmonellae could be detected and the recent institution in Britain of compulsory pasteurization of liquid egg are examples. Much has been written concerning the destruction of salmonellae in egg products, and such processes as heating, acidification and heating, and alkalinization have been recommended. That it is possible to free egg products of salmonellae is attested to by the British experience with frozen whole egg imported from Poland. This product, which previously had been heavily contaminated, was completely free of salmonellae in 1955 and 1956. In conversations with persons familiar with the technical aspects of treatment, the impression is gained that from a practical and technological standpoint there still is much to be desired in the treatment of egg products to free them of salmonellae. Certainly the long heating periods applied to dried albumin are most cumbersome. It seems likely that more practical methods of treatment will be devised. Radiation is being actively investigated and may eventually aid in the solution of the problem.

The foregoing remarks are intended to emphasize the frequency with which salmonellae occur in human foods, and particularly foods of animal origin. In this connection, two observations by Cockburn (1) should be cited. The first is that in his experience it was possible to identify the food responsible for the infection in only 12 percent of the outbreaks investigated and that from this small percentage one was not justified in drawing sweeping conclusions regarding foods responsible for the remaining 88 percent of the epidemics. This statement was not made to de-emphasize the importance of food-borne salmonellosis, but rather to emphasize the difficulties encountered in its epidemiologic investigation. The second observation by Cockburn which should be mentioned is that it is not invariably an originally contaminated item of food which gives rise to infection. Organisms may be transferred from food to food through soilage of utensils or, as Taylor (2) has shown, through the production of human carriers. These observations indicate that it is not only the particular item of contaminated food which is of importance but that the introduction of salmonellae into the area of food preparation by any method should be avoided insofar as possible.

Recently, in the United States there has been a revival of interest in epidemics of salmonellosis in hospitals, due largely to the experience with *S. derby* in the northeastern region. Previously in this country, the hospital epidemics that have been most publicized, in contrast to the *S. derby* situation, largely have involved infants in nurseries or pediatric wards. To illustrate that hospital outbreaks are by no means unusual, reference again is made to the data collected in England and Wales (3). In Table 4 are listed the total numbers of outbreaks of salmonellosis and staphylococcic food poisoning for which locations were specified. Also listed are the outbreaks that occurred in hospitals. Almost half of the outbreaks of salmonellosis were listed as occurring in hospitals, illustrating the frequency of such occurrences. Perhaps this should not be unexpected, since the population at risk is composed largely of the very young and the infirm in whom there is a high degree of susceptibility. On the contrary, less than 10 percent of the outbreaks of staphylococcus food poisoning reported by location occurred in hospitals, indicating that lack of refrigeration and mishandling of foods probably were not responsible for the high proportion of hospital outbreaks of salmonellosis. Whether the character of the hospital diet, compared to that of the general population, accounts for the high proportion of hospital outbreaks is a question that warrants investigation.

Table 4.–*Reported locations of outbreaks England and Wales*

Year	Salmonellosis		Staphylococcic Food Poisoning	
	Total	Hospitals	Total	Hospitals
1953	87	40	37	2
1954	66	36	45	3
1955	66	39	49	3
1956	65	33	44	8
1957	56	28	29	2
1958	52	23	22	5
1959	57	28	23	0
1960	52	25	28	1
1961	47	13	22	4
1962	44	15	14	0
Total	592	280 (47.3%)	313	28 (8.9%)

Nowhere in the program of the conference was the topic of the human carrier and his role in the production of salmonellosis mentioned. Nevertheless, it is felt that some mention must be made of the problem. This is not a popular subject for discussion because of the difficulty of its solution. It once was believed that salmonellosis was a self-limiting disease in which the infecting organisms disappeared from the intestinal tract shortly after the disappearance of symptoms. Today, due to more efficient isolation methods and closer surveillance of convalescent patients, it is known that a certain proportion of persons affected with salmonellosis may continue to excrete the organisms for months or years and that this excretion may or may not be accompanied by intermittent bouts of diarrhea. Administration of broad spectrum antibiotics largely is ineffectual in terminating the carrier state. The carrier state exists not only in convalescents but also in persons without history of overt infection. In the more highly developed countries, the carrier rate among asymptomatic persons has been estimated at levels ranging from 0.2 percent to 5.0 percent. In developing regions, even higher rates have been reported.

In the United States, fecal examination of food handlers now is not done as generally as in the past. This change in practice is quite understandable when one considers that the procedure originally was designed to detect typhoid carriers and that very few such persons now remain within the age brackets likely to be employed as food handlers. The work has become extremely unproductive insofar as detection of typhoid carriers is concerned; and, in order to detect carriers of other salmonella serotypes in a manner at all approaching efficiency, the examinations would necessarily have to be done very frequently because of the transient nature of the carrier state in the majority of such persons. That food often is contaminated by human carriers is evidenced by the isolation of such host-adapted forms as S. typhi, S. paratyphi A, S. paratyphi B, and shigellae from such products as meats, frozen eggs, and coconut. In our own experience, instances have occurred in which previously detected carriers of salmonellae employed as food handlers have given rise to epidemics. In addition, catering firms operating over a large area have been involved in the transmission of salmonellosis due to the same serotype in widely separated locations in different States. These occurrences were spaced over a period of months; and, when the food handlers of the firms involved finally were examined, many were found to be carriers. It must be admitted that examination of food handlers in the retrospective investigation of epidemics fails to distinguish culprits and victims; and, in many cases, the high proportion of carriers found indicates that only additional inapparent cases are being detected. Nevertheless, unequivocal evidence of the role of the human carrier in the transmission of salmonellosis is sometimes obtained, as in the instances just cited. Due to handling of large numbers of contaminated food items and frequent ingestion of small numbers of the organisms, it seems very likely that the carrier state is an occupational hazard of food handlers. Forty-seven percent of 56 outbreaks reviewed by Felsenfeld and Young were attributed to human carriers. These figures include only outbreaks due to non-host-adapted types, S. typhi, S. paratyphi A, and S. paratyphi B being excluded.

Since it is not practical to examine all food handlers at frequent intervals to detect temporary carriers of salmonellae, it becomes mandatory to instruct all such employees in the elements of personal hygiene and to insist on strict enforcement of these principles. Obviously, persons affected with any form of gastrointestinal disturbance should not be permitted to handle food until it is established that they are not excreting enteric pathogens. Inasmuch as salmonellosis is known to occur so frequently in hospitals and geriatric institutions and since the populations involved are more susceptible than the public at large, it would be most desirable to require frequent and regular fecal examinations of food handlers in these locations. It is ironic that, in some localities, fecal examination of food handlers in restaurants has been mandatory; whereas there was no such requirement for hospital employees.

Finally, we come to the problem of the presence of salmonellae in animal feeds and feed ingredients. Choice of this subject as the last topic of discussion was deliberate, since it is felt that here the solution of the problem of salmonellosis should begin. Obviously, any cycle of infection should be attacked at all vulnerable points. So long as we continue deliberately to seed our animal population, we are contributing to the maintenance of the present situation of numerous cases and carriers of salmonellosis in animals; to contamination of holding lots, abattoirs, processing plants, carcasses, and meats; to the presence of salmonellae in egg products and food mixes; to the production of human carriers; and to the continuance of the problem of salmonellosis as it exists today.

That salmonellae occur frequently in feed and feed ingredients is attested by the surveys conducted by the Public Health Laboratory Service in England and Wales and the U. S. Department of Agriculture in this country, as well as by the published findings of numerous individual investigators. Probably the most spectacular report among all these investigations was that of Harvey and Price (4), who isolated 17 distinct salmonella serotypes from one specimen of crushed bone. A comparison was made of the serotypes isolated from man, domestic mammals, fowls, eggs and egg products, water and sewage, and feed ingredients. Cultures isolated over a period of years in the United States, Britain, and the Netherlands were summarized and compared separately. The individual serotypes from each of the sources were arranged numerically, and the numerical ratings of the serotypes from each of the other sources compared with them. Within each country, there was rather remarkable agreement in the incidence of serotypes isolated from all sources other than feed and feed ingredients. On the contrary, the numerical incidence of the individual serotypes isolated from feed and feed ingredients was markedly different from the incidence of those derived from other sources. This is by no means a new observation and other persons have remarked on this discrepancy. For example, S. typhi-murium is by far the most frequently encountered serotype in overt infections of man and animals, but it has been found less frequently in feed and feed ingredients than have several other serotypes. The discrepancy in the serotypes found in feeds and in animals probably has contributed more than any other circumstance to lack of more positive action being taken to eliminate salmonellae from feeds. Three published observations indicate that this discrepancy is more apparent than real. First, Smith (5) called attention to the fact that methods used in the isolation of salmonellae had a marked influence on the types recovered. While Smith referred in particular to S. cholerae-suis, we have little or no knowledge of the differential effects of commonly used isolation methods on individual serotypes in mixed populations of salmonellae. Second, Kampelmacher and his associates (6) undertook a very intensive examination of fish meal imported into the Netherlands. As a result of this intensive search, S. typhi-murium emerged as the predominant serotype, occurring almost three times as often as the next most frequently found type. Third, Henderson (7) fed chicks graded doses of seven commonly occurring salmonella serotypes including S. typhi-murium. The pen mortality in the chicks fed 1 to 10 cells of S. typhi-murium greatly exceeded that produced by feeding 100 million cells of any of the other six serotypes. Further, mortality among all chicks fed S. typhi-murium was more than four times as great as that in chicks fed similar doses of the next most fatal serotype. Thus, when one considers that we know very little about the differential effects of our isolation methods on different serotypes, that really adequate sampling, in some instances at least, reveals a much higher incidence of S. typhimurium in feed ingredients than previously was suspected, and that S. typhimurium produces a higher mortality than other serotypes, the discrepancy between the incidence of serotypes in feeds and overt salmonellosis is explained. No longer can we take comfort from the fact that the salmonellae found in feeds are not those usually reported in salmonellosis.

The mandatory sterilization of imported feed stuffs in several European countries indicates the concern with which the problem is viewed in some quarters. Considering the high incidence of salmonellosis in some central European countries, it is, perhaps, rather naive to require sterilization only of imported materials. For some years, manufacturers of human foods have been seeking ingredients in which no salmonellae could be demonstrated. With the growing awareness of the consumer of the possibilities of transmission of salmonellosis to flocks and herds through feed stuffs, it is quite probable that feed manufacturers also will seek ingredients which do not contain the organisms. Such requirements together with proper safeguards against contamination during processing, packaging, transportation, and storage should result in marked improvement of the present situation and permit us to go forward in the control of salmonellosis with a much greater degree of confidence.

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Salmonella Surveillance Program in the United States

Philip S. Brachman, M.D.[1] *and*
James B. Goldsby, M.S.[2]

In November 1961, following a report from Canada concerning studies of Salmonella thompson gastroenteritis associated with commercial cake mixes, the Communicable Disease Center initiated investigations of S. thompson isolations reported from the Enteric Bacteriology Laboratory of the Communicable Disease Center. A focus of 13 cases in Detroit, Michigan, resulted in specific investigations by the Michigan and Detroit health departments and Dr. Eli A. Friedman, former Epidemic Intelligence Service Officer. An association of these cases with commercial eggs was clearly indicated. These studies led the Communicable Disease Center to initiate a surveillance program among a few selected interested States in April 1962. During the following months, additional States joined the program. Subsequently, the State and Territorial Laboratory Directors and Epidemiologists recognized the need for a nationwide surveillance of salmonellosis and requested the Communicable Disease Center to develop a Salmonella Surveillance Program, a report of which is the basis of this presentation. In January 1963, surveillance was expanded to include all 50 States, Puerto Rico, the Virgin Islands, New York City, and the Beth Israel Salmonella Laboratory in New York City.

The aim of our surveillance program has been to design a reporting system that would collect data, giving a clearer picture, a truer definition of the salmonella problems in the United States. Vital to the program has been the need for it to be sensitive enough to develop the endemic patterns and yet acute enough to allow for the rapid identification of an epidemic or a potential epidemic, especially one with interstate involvement.

The nationwide surveillance program has served to focus more attention on the endemic problems, by State and local health officials, and has extended to the private practitioners who, in some instances, have been stimulated to obtain a greater number of cultures from patients with diarrhea. Additionally, the surveillance program has allowed us to increase our competence in the area of salmonellosis, so we are more qualified and effective in providing epidemic aid assistance.

We also have appreciated the need to disseminate information collected from the States, so that a routine report available to all participating and interested people was incorporated as an integral part of the program.

The basis for our surveillance program is a weekly report, mailed on Friday afternoons from the reporting centers, summarizing the salmonella isolates identified during the preceding 7 days in the respective laboratories. Isolates are either from those clinically ill or from carriers. A typical reporting form (Figure 1) lists the individual serotype identified, and name of patient, initials, or laboratory number, so that duplicate cultures in the same or different laboratories can be excluded, and also in order that the percent of isolates that are family related can be determined. The sex and age are given so that age specific attack rates can be calculated. The county of residence at time of isolation is requested in order to pinpoint focal outbreaks in adjacent counties in two States.

Some States will process specimens from another State, so origin (of patient), if other than reporting State, is also noted. At the bottom, nonhuman isolations are reported by serotype, source, and county. All data included on each individual provide basic keys to the epidemiology of salmonellosis.

Additionally, the U. S. Department of Agriculture, National Animal Disease Laboratory at Ames, Iowa, sends us regular reports of their nonhuman isolates. Dr. Van Houweling will discuss this later this morning.

The reporting form is a five-copy, snap-out form, with carbon paper between each sheet. The last two copies are kept in the State, one copy remains with the department that filled out the form, the other copy goes to the laboratory director or epidem-

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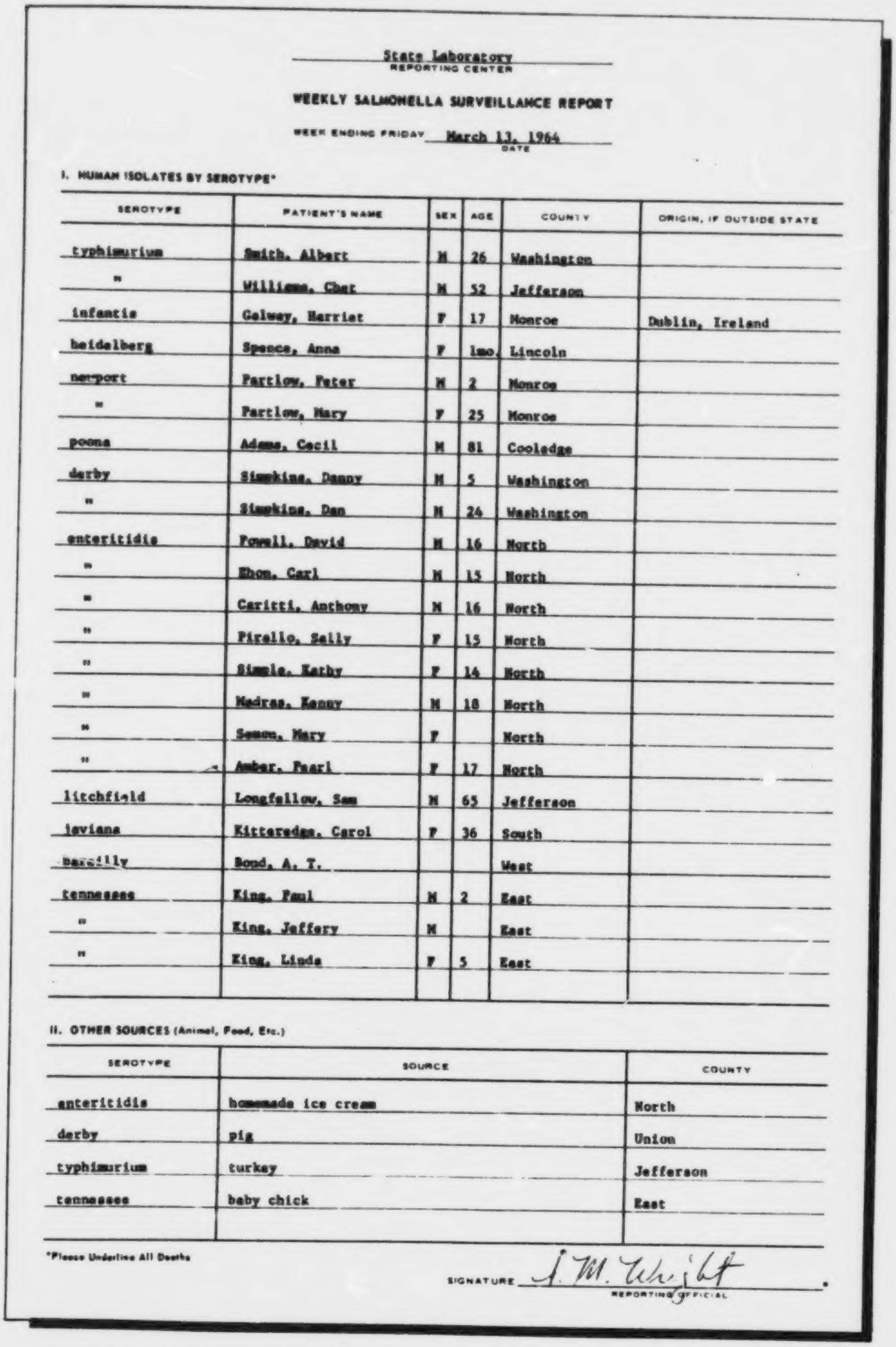
[2] Statistician, Investigations Section, Epidemiology Branch, Communicable Disease Center, Atlanta, Georgia.

Figure 1.–*Weekly Salmonella Surveillance Report*



State Laboratory
REPORTING CENTER

WEEKLY SALMONELLA SURVEILLANCE REPORT

WEEK ENDING FRIDAY March 13, 1964
DATE

I. HUMAN ISOLATES BY SEROTYPE*

SEROTYPE	PATIENT'S NAME	SEX	AGE	COUNTY	ORIGIN, IF OUTSIDE STATE
typhimurium	Smith, Albert	M	26	Washington	
"	Williams, Chet	M	52	Jefferson	
infantis	Galway, Harriet	F	17	Monroe	Dublin, Ireland
heidelberg	Spence, Anna	F	1mo.	Lincoln	
newport	Partlow, Peter	M	2	Monroe	
"	Partlow, Mary	F	25	Monroe	
poona	Adams, Cecil	M	81	Cooledge	
derby	Simpkins, Danny	M	5	Washington	
"	Simpkins, Dan	M	24	Washington	
enteritidis	Powell, David	M	16	North	
"	Ebon, Carl	M	15	North	
"	Caritti, Anthony	M	16	North	
"	Pirello, Sally	F	15	North	
"	Simple, Kerby	F	14	North	
"	Madras, Kenny	M	18	North	
"	Semon, Mary	F		North	
"	Amber, Pearl	F	17	North	
litchfield	Longfellow, Sam	M	65	Jefferson	
javiana	Kitteredge, Carol	F	36	South	
bareilly	Bond, A. T.			West	
tennessee	King, Paul	M	2	East	
"	King, Jeffery	M		East	
"	King, Linda	F	5	East	

II. OTHER SOURCES (Animal, Food, Etc.)

SEROTYPE	SOURCE	COUNTY
enteritidis	homemade ice cream	North
derby	pig	Union
typhimurium	turkey	Jefferson
tennessee	baby chick	East

*Please Underline All Deaths

SIGNATURE J. M. Wright.
REPORTING OFFICIAL

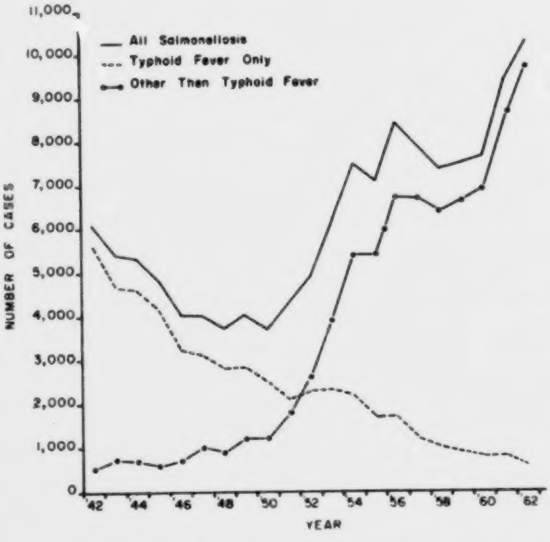
iologist, as the case may be. The other three copies, which we keep, are distributed to (1) the epidemiologist for his scrutiny; (2) the statistician for the running record; (3) the IBM punch card section, so that all information pertaining to each isolate can be punched onto an IBM card.

Upon receipt of the individual State reports, the data is entered on our salmonella board, as Dr. McCall will discuss later. In examining the reports, we are immediately alert to two potential findings; one is the increased number of isolates of the same serotype from any one or several States. Secondly, we look for the isolation of infrequently or rarely isolated serotypes. The occurrence of these serves as a radioactive marker—potentially making it possible to trace the pathway of infection from victim through vehicle to source.

Each month these data are organized into a report, copies of which have been given to each of you. The monthly report not only summarizes isolations from both human and nonhuman sources on a national scale, but also reports on field and laboratory studies conducted by the States or by the CDC, brings in pertinent industrial and international information, and reviews interesting articles in the literature.

In Figure 2, we have reviewed the annual data reported from the States to the National Office of Vital Statistics (of the Public Health Service) from 1942 through 1959, and since 1960 to the CDC. It can be seen that in 1942, out of 6,000 salmonella isolates reported, 5,600 (or 92 percent) were S. typhi; whereas, only 500 other salmonella were identified. Gradually and steadily the number of S. typhi has declined, so that in 1962, only 600 (or 6 percent) of the total isolates reported were this serotype; whereas, other salmonella isolated increased to 9,700. Any comparison between these data and those which we have collected during 1963 must be tempered by the fact that the methods of data collection have been different. Nevertheless, in 1963, a total of 18,649 human isolates of salmonella were reported to CDC from the States. Of the total, 712, or 3.8 percent, were S. typhi.

Figure 2.–*Reported incidence of human salmonellosis (United States, 1942-62)*



Source: MMWR Annual Supplements, 1955, 1963.

Table 1.–*The 10 salmonella serotypes most frequently reported as isolated from human specimens United States–1963*
[Total 1963 salmonella isolations - 18,649]

Rank	Serotype	Number	Percent of 1963 total
1	*typhi-murium*	5,435	29.1
2	*derby*	1,610	8.6
3	*heidelberg*	1,533	8.2
4	*newport*	1,080	5.8
5	*infantis*	970	5.2
6	*enteritidis*	801	4.3
7	*typhi*	706	3.8
8	*saint-paul*	586	3.1
9	*oranienburg*	539	2.9
10	*montevideo*	490	2.6
	Total	13,750	73.7

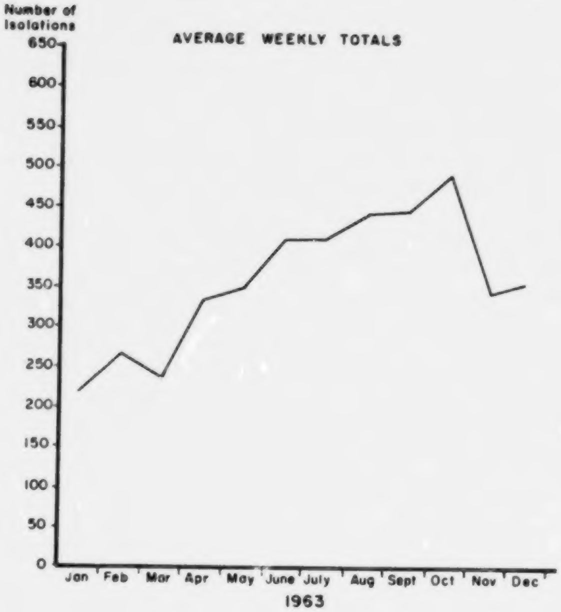
The 10 most frequently isolated serotypes, which represent 8.3 percent of the total (121) different serotypes isolated, accounted for 73.6 percent of the total organisms reported for 1963 (Table 1). S. typhi-murium headed the list every month, and accounted for 29.1 percent of the total isolates. Other serotypes that have appeared on the "popularity list" every month include S. heidelberg. S. newport, and S. infantis. S. derby was the second most frequent isolate for 1963, with 8.6 percent of the total; this represents the intensively studied, interstate outbreak that inundated the northeastern States, beginning in April 1963.

Surveillance reports from other countries have shown that S. typhi-murium has been universally the most commonly identified serotype. The next most frequently isolated serotypes have varied widely, indicating the effect of local factors on the prevalence and spread of salmonella. Examination of nonhuman isolates reveals similarities in the most commonly isolated serotypes, as Dr. Van Houweling will show.

Among the total isolates, 3,126 or 16.7 percent were from among members of families in which there were familial outbreaks.

The average weekly totals plotted by month show a gradual increase in isolates from January to October, with a marked fall in November, followed by a slight rise in December (Figure 3). At first glance, the steady increase in reported isolates was felt possibly to represent either (1) improve-

Figure 3.–*Reported human isolations of salmonellae in the United States*



NOTE: Average weekly totals rather than monthly totals are presented because some months have 5 instead of 4 weeks.

ments in reporting, or (2) an increased number of cultures being obtained, or (3) a seasonal prevalence of salmonella isolations. When the monthly data from 1942-1962 are examined, essentially the same type of monthly distribution is seen, with the lowest number of isolates reported in January or February, and the peak in August, followed by a gradual decline. These data represent cases of salmonellosis, rather than isolations of salmonellae. Therefore, the dip in November and December was expected earlier, around September or October, near the end of the summer "vacation and communal-gathering season." This could be what actually occurred in 1963, since indications are that there was a lag of as much as 6 to 8 weeks between the onset of cases and the reporting of isolates.

Figure 4 gives the estimated age specific attack rates for isolations of salmonella for 1963, utilizing estimated population data for 1962. The highest attack rate occurred in children less than 1 year of age, 43 isolates per 100,000 children. The 1 to 4-year group had an isolation rate of 17 per 100,000, and all other groups, 7 or less per 100,000.

The high rate for those less than 1 year of age probably represents a combination of influencing factors; one is their reported increased susceptibility due to immaturity of defense mechanisms. Another important factor is that a child with diarrhea is more likely to be seen by a physician than an adult is and rectal swabs are more likely to be obtained for cultures. Many studies have clearly indicated that a greater number of cases are diagnosed and reported when additional cultures are taken.

In the same vein, the increased susceptibility of the elderly has been reported. Our data show a gradual increase in the rate of isolation from age group 30 to 39 years (3.5 isolations per 100,000) up to 80 plus years (6.5 isolations per 100,000). Data compiled during 1963 show a death rate of 0.33 percent. This is a minimal figure, since State health departments do not always receive this information at the time cultures are sent in from the local levels.

The manner in which surveillance can function as an early alarm system for an epidemic will be discussed by Dr. McCall.

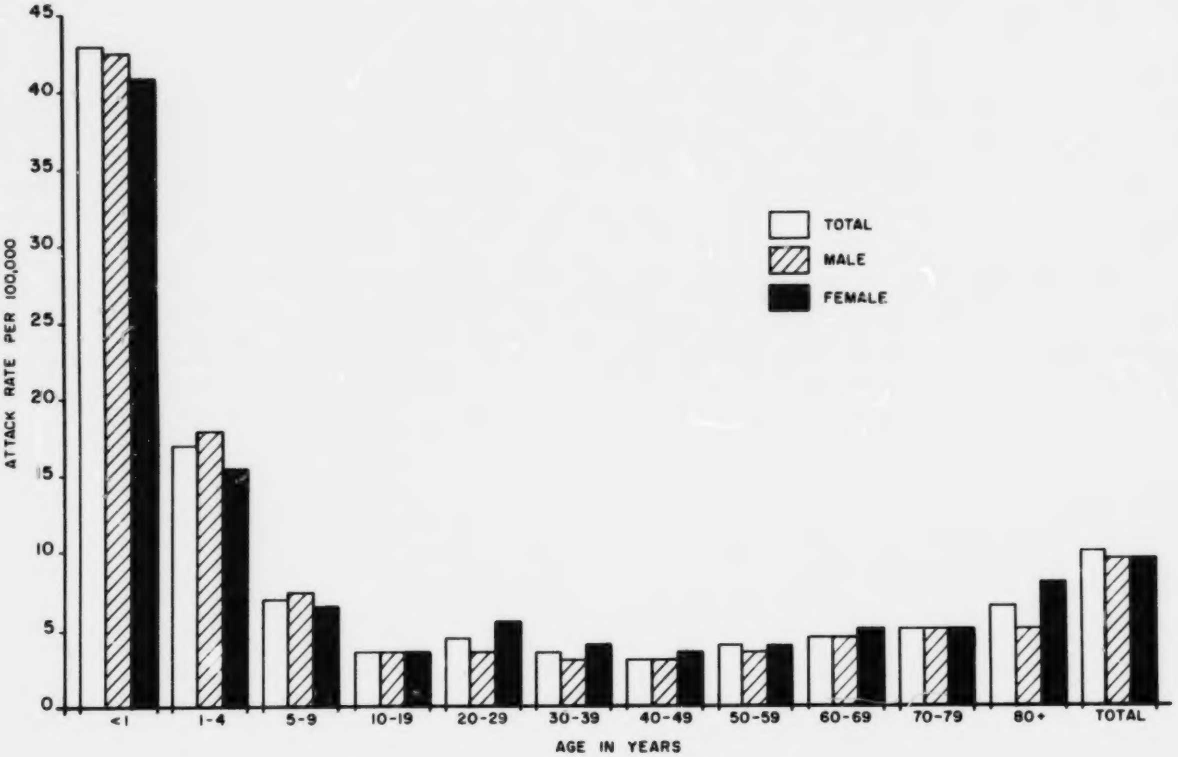
We have recorded, as others have, the regionalization of certain serotypes of salmonella. Thus, S. cubana has been isolated 40 times in 1963, and all isolations have been reported from States east of the Mississippi, with the exception of one isolation from Missouri; S. miami has been reported 65 times, and 86 percent have been from Florida; S. atlanta has been isolated only 11 times, and all isolations occurred in Georgia; S. saphra has been isolated 5 times, always from children under 5 years of age living on the Gulf of Mexico (Texas 1, Louisiana 3, Florida 1); S. loma-linda has occurred 6 times, 5 times from people associated with Loma Linda College in California.

These data give the impression that the source of infection in these instances is localized and does not have the opportunity for wide dissemination. This may indicate that the vehicle of infection is not distributed beyond the local area, or that the organisms are not able to survive distant transportation or transplantation, or that the infection dose in the vehicle is low, and, with normal attribution, decreases below clinical infection levels within a short time.

Surveillance has also shown that some serotypes, initially isolated relatively infrequently, can assume national importance as in a common-source interstate epidemic, and then return to their former rarity, such as S. hartford, while others remain at a high rate of isolations, such as S. reading. In both of these studies, the vehicle of infection was not identified. Reasons for these differences are not clear, but may be related to the vehicle of infection, degree of contamination, virulence of the organism, or other undefined factors. Isolations of these two serotypes from nonhuman sources may explain the differences in persistence as a human isolate today. S. reading is commonly identified from nonhuman sources (poultry is its most common source); whereas S. hartford remains an infrequent nonhuman isolate.

As methods of control are recognized and instituted, surveillance enters another phase of activity. Utilizing background data developed during the first year of surveillance, we can evaluate the

Figure 4.–*Estimated attack rates of salmonellosis in the United States, 1963*[1]



[1] Number of human isolations of salmonellae during 1963, per 100,000 estimated U.S. population, July 1, 1962.

effectiveness of control methods. Laboratory surveillance, that is, the routine culturing of specific food and feed products, can be a distinct aid in surveillance. Though we do not know the importance of the actual level of contamination, laboratory surveillance can be used as a guide, even a warning system for potential trouble. The routine culturing of foodstuffs for salmonella content should be a part of a national surveillance program.

In summary, the Salmonella Surveillance program will only be as effective as the sponsoring State health departments will allow it to be. Since the number of isolates reported for 1963 probably represents only a small fraction of the true incidence figure, there is need for increased local effort to identify all cases and carriers. This will be necessary if the total scope of the problem is to be brought to the surface.

The blanket of surveillance must be broad enough to cover all areas that contribute to salmonellosis as a public health problem. Any hole will allow the most elusive member to escape—and he may be the keystone in the structure of control.

Surveillance is not a panacea of control, but if properly conceived and if accepted, it can be not only a beacon light, an alarm system, and a chronicler of events, but also a guide to better things for better living through surveillance.

Salmonellosis in the United Kingdom

Joan Taylor, M.B.[1]

Salmonellosis has been recognized as a problem in Great Britain since the beginning of the present century. After the First World War, Savage, White, and others investigated a number of outbreaks which led to the recognition of the salmonella group as the main cause of food poisoning and clearly showed that protein breakdown products, such as ptomaines, could no longer be held responsible for the condition. Between the wars, and for some years after the Second World War, it was believed that the majority of human outbreaks could be traced to the infection of food by human carriers; although at the same time, it was well recognized that domestic animals also suffered from salmonellosis. Nevertheless, the human carrier received most of the blame, and little attempt was made to track down more remote sources.

The first important change came during the last war when a collaborative study of salmonellae in imported dried egg was carried out in a number of laboratories in different parts of the United Kingdom, and serotypes hitherto unknown in the United Kingdom were isolated from imported dried egg and from human infection. This drew attention to the effect of imported contaminated human food on human salmonellosis.

In 1949, the reporting of food poisoning incidents was put on a systematic basis by the issue of a standard report form to Medical Officers of Health, who since that year have made annual returns. In addition, hospital pathologists and public health bacteriologists have reported cases and incidents so that since 1955 it is probable that most incidents in England and Wales have been recorded; nevertheless, a number of sporadic cases are still not investigated, so are not recognized.

In the Kauffmann-White table, there are about 900 salmonella serotypes of which 99 have been isolated on more than one occasion from human infection during the 5-year period 1958 to 1962 (1-5 and Dr. E. S. Anderson, pers. com.), Table 1. The frequency of Salmonella typhi and the 10 most common serotypes is shown in Table 2. Of these 10 common types, 7 have occurred regularly year after year and account for 87 percent of human salmonellosis. Although these types have turned up so consistently, there is often a marked fluctuation in the number of isolations from one year to another. For instance, 516 cases or incidents of S. paratyphi B infection were noted in 1959; whereas, in 1962, there were only 197. Types such as S. montevideo and others rarely occur in this common group or may show a waxing and waning incidence followed by almost or complete absence.

Many factors play a part in creating such a picture. Those types which are consistently in the top 10 come from sources which are distributed widely and regularly throughout the United Kingdom, such as human food originating from home-produced animals. These sources constitute a continuing source of infection. Some imported foods also come into this category, such as frozen egg imported prior to 1964. Tables 1 and 2 also give many examples of serotypes which occur rarely or which have been important during one particular year, and then have disappeared. Such types, obviously, are not widely distributed and do not have constant sources.

Referring to previous reports (6-13), S. typhi-murium, which was responsible for 1,300 incidents in 1949, increased in importance and by 1955 caused a maximum of 4,200 incidents. The number of incidents due to this type remained steady at about 3,000 from 1956 to 1959; thereafter, the number fell, so that in 1962 only 1,864 were recorded. Incidents due to other serotypes excluding S. paratyphi B, remained fairly steady at about 500 annually from 1949 to 1954, then increased to a maximum of 1,800 in 1959, but by 1962 attained a lower level of 982 incidents. Figure 1 shows these changes. The main increase in S. typhi-murium infection, which started about 1952, coincided with a change in abattoir regulations when private abattoirs were allowed to operate; the increase in infection with other serotypes began about 1955.

Reports from 1949 to 1962 give the number of general outbreaks, family outbreaks, and sporadic cases recorded in the United Kingdom. These are summarized in Table 3. The annual incidence of general outbreaks due to S. typhi-murium has declined sharply from 114 to 43, but the number of general outbreaks due to other serotypes has fallen more gradually from 41 to 29. The annual number

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[1] Salmonella Reference Laboratory, Central Public Health Laboratory, Public Health Laboratory Service, London, England.

Table 1.—*List of salmonella serotypes from man in England and Wales (1958-62)*[1]

Serotype	Number					Serotype	Number				
	1958	1959	1960	1961	1962		1958	1959	1960	1961	1962
typhi-murium	3,406	3,241	2,943	2,544	1,864	*kottbus*	...	6	6	...	2
abadina	...	...	2	...	...	*kotte*	...	...	...	2	...
aberdeen	...	11	...	3	...	*lexington*	...	...	7	6	1
abony	1	11	...	5	2	*limete*	...	4	...	...	1
adelaide	1	5	2	3	1	*litchfield*	...	...	...	2	...
africana	2	...	...	...	...	*livingstone*	2	1	4	2	5
agama	1	4	2	9	2	*london*	2	2	1	4	1
anatum	55	54	17	31	19	*manchester*	4	11	5	2	1
bareilly	32	26	18	14	8	*manhattan*	8	9	2	44	7
binza	...	...	...	2	...	*meleagridis*	4	13	19	56	9
bispebjerg	...	...	2	...	1	*menston*	...	...	25	45	33
bleadon	...	1	...	1	...	*mikawasima*	...	1	2	2	...
blockley	1	41	9	7	4	*minnesota*	...	...	2	1	...
bovis morbificans	27	40	30	16	11	*montevideo*	5	43	12	19	11
bradford	...	...	1	...	1	*muenchen*	31	42	7	3	2
braenderup	2	2	...	2	1	*nagoya*	...	...	...	3	5
brancaster	...	1	...	...	1	*new-brunswick*	4	...	...	...	...
brandenburg	7	6	39	53	7	*newington*	11	2	1	6	1
bredeney	50	41	3	14	103	*newport*	118	319	54	61	64
butantan	4	...	5	...	...	*oranienburg*	8	15	8	1	5
california	1	1	1	...	...	*orion*	8	2	1	1	...
chailey	1	1	3	...	...	*panama*	5	6	9	7	8
chester	20	14	4	6	3	*paratyphi B*[2]	342	516	350	423	197
chicago	...	...	...	2	...	*pomona*	...	...	...	...	2
cholerae-suis	11	24	10	4	4	*poona*	...	4	7	...	3
coeln	...	...	...	6	...	*potsdam*	2	12	4	...	3
cubana	...	1	...	3	...	*pullorum*	...	2	1	...	1
derby	58	31	13	8	11	*reading*	15	7	6	8	31
dublin	30	36	27	24	10	*richmond*	...	...	1	2	...
durham	2	1	...	...	4	*rubislaw*	...	...	...	...	2
ealing	...	...	3	1	...	*saint-paul*	95	83	42	69	32
eastbourne	2	1	...	...	1	*san-diego*	...	...	...	2	...
enteritidis	113	119	145	90	93	*schwarzengrund*	35	27	8	3	2
gaminara	2	...	...	...	...	*senftenberg*	10	13	11	8	4
give	4	25	9	10	8	*stanley*	6	11	68	42	50
glostrup	...	...	...	2	4	*stanleyville*	1	...	2	2	...
haifa	1	...	...	...	1	*stourbridge*	...	...	...	3	...
havana	...	1	1	1	1	*takoradi*	1	...	1	3	...
heidelberg	308	182	118	289	133	*taksony*	1	1	1	1	...
huvudsta	...	...	3	...	...	*tennessee*	...	16	9	1	10
hvittingfoss	1	...	1	...	1	*thompson*	132	217	122	54	39
ibadan	1	...	1	...	...	*typhi*[2]	141	154	119	122	171
idikan	6	2	...	...	...	*vejle*	...	...	...	5	1
infantis	18	57	28	2	12	*virchow*	...	...	7	5	2
irumu	1	...	2	1	2	*wangata*	15	1	...	6	1
javiana	...	1	...	1	...	*waycross*	...	...	1	2	1
johannesburg	7	...	1	...	4	*weltevreden*	...	...	2	2	2
kaapstad	...	4	...	1	...	*westerstede*	...	...	...	...	2
kentucky	1	8	...	6	...	*worthington*	23	4	3	4	3
kiambu	3	6	8	1	3						
						Total	5,435	5,802	5,474	4,400	3,214

[1] Omitting serotypes which appeared once only in the 5-year period.

[2] Figures kindly supplied by Dr. E. S. Anderson.

Table 2.—Salmonella typhi *and the 10 most common salmonella serotypes in man (1958-62)*

[Figures in parentheses give order of frequency in each year]

Serotype	Number of isolations				
	1958	1959	1960	1951	1962
typhi	141	154	119	122	171
anatum	55 (9)	54 (9)	....	....	....
brandenburg	....	....	39 (8)	53 (9)	....
bredeney	50 (10)	....	....	....	103 (4)
derby	58 (8)	....	....	....	....
enteritidis	113 (6)	119 (5)	145 (3)	90 (4)	93 (5)
heidelberg	308 (3)	182 (5)	118 (5)	289 (3)	133 (3)
infantis	....	57 (8)	28 (10)	....	....
meleagridis	....	....	....	56 (7)	....
menston	....	....	....	45 (10)	33 (9)
montevideo	....	43 (10)	....	....	....
newport	118 (5)	319 (3)	54 (7)	61 (6)	64 (6)
paratyphi B	342 (2)	516 (2)	350 (2)	423 (2)	197 (2)
saint-paul	95 (7)	83 (7)	42 (8)	69 (5)	32 (10)
stanley	....	....	68 (6)	....	50 (7)
thompson	132 (4)	207 (4)	122 (4)	54 (8)	39 (8)
typhi-murium	3,406 (1)	3,241 (1)	2,943 (1)	2,544 (1)	1,864 (1)

Table 3.—*Average annual incidents, 1949-62*

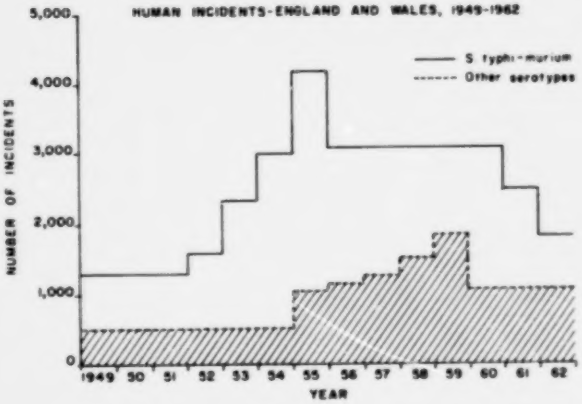
	General outbreaks	Family outbreaks	Sporadic cases		General outbreaks	Family outbreaks	Sporadic cases
typhi-murium				Other serotypes			
1949-54	114	224	1,463	1949-54	41	52	403
1955-59	63	279	3,025	1955-59	38	113	1,236
1960-62	43	219	2,163	1960-62	29	104	966

Table 4.—*Seven common human salmonella serotypes in some human foods*

Serotype	Coconut (1959-62)	Egg products (1956-62)						Pie meat, sausages (1960-62)
		United States	Argentina	Australia	China	The Netherlands	United Kingdom	
typhi-murium	54	27	20	1,059	153	41	513	55
enteritidis	....	.....	....	1	9	....	......	....
heidelberg	1	15	....	....	1	1	......	25
newport	31	6	6	2	42	....	......	2
paratyphi B	200	.....	....	....	216	....	......	4
saint-paul	....	.....	....	....	....	....	......	....
thompson	17	.....	....	11	1,230	4	60	....
Percent of total represented by 7 common serotypes	19	10	39	70	74	19	87	32
Number of strains	1,618	490	67	1,541	2,225	244	659	265
Number of serotypes	69	29	10	27	34	14	10	32

of family outbreaks due to S. typhi-murium rose slightly between 1955 and 1959, then resumed its former level. Family outbreaks due to other serotypes doubled in number in the 1955 to 1959 period and have remained at this level. The number of sporadic cases due to S. typhi-murium and to other serotypes increased sharply between 1955 and 1959, then diminished, but remained at a level exceeding that of the 1949 to 1954 period. The reduction in incidents was greatest for general outbreaks and least for family outbreaks and sporadic cases. This may reflect hygienic advances in large-scale food preparation.

Figure 1.–*Outbreaks, family outbreaks, sporadic cases*



SEROTYPES IN HUMAN FOOD

The most common sources of salmonellae are the animal protein foods. Except from processed coconut (14), rarely have salmonella organisms been isolated from vegetables or fats. The frequency distribution of the seven common human types in some human foods is shown in Table 4, but it must be emphasized that infection rate is not given. Generally speaking, many samples of coconut and Chinese egg are infected; whereas, the infection rate is low in many of the other products listed.

The amount of coconut consumed in this country is negligible when compared with the amount of egg and egg products; nevertheless, some outbreaks of S. paratyphi B (15) have been traced to the consumption of coconut. Many outbreaks of S. paratyphi B (16) and some of S. thompson (17) were traced to imported Chinese egg products, and some outbreaks of S. typhi-murium infection have been traced to frozen whole egg produced in the United Kingdom. Table 4 is of particular interest in emphasizing the fact that the highest percentage of the common human serotypes is found in home-produced material. S. paratyphi B has never been isolated from home-produced egg products, and the view generally held is that the presence of this organism suggests contamination directly or indirectly by human feces. Such contamination can come from the food handlers, or be transmitted by contaminated water used in the food processing factories, and by flies.

The salmonellae found in foods produced by and processed in the developed countries are the same serotypes most commonly isolated from the animals from which the food is produced. The distribution of the seven common human types has been emphasized, but occasionally the foods shown in Table 4 cause outbreaks due to rare types such as S. irumu. In this case, contaminated South African frozen egg caused an outbreak affecting 10 people (18).

In 1964, new regulations came into force, and now all mixed whole egg, whether imported or home-produced, must be pasteurized. It will be interesting to note the effect on the incidence of human salmonellosis.

SALMONELLA IN DOMESTIC ANIMALS

Table 5 shows the types and number of salmonellae isolated from domestic animals from 1956 to 1961. These strains were identified in the Salmonella Reference Laboratory, which is likely to receive the more unusual types and less likely to be sent the truly host-specific types such as S. pullorum from poultry or S. cholerae-suis from swine; such strains when isolated from other animals, are sent for confirmation.

Table 6 gives the frequency distribution in animals in the United Kingdom of the seven common human salmonella types and indicates the sources in the United Kingdom from which it is likely that human infection originated. Home-produced meat from bovines and pigs is probably the source of much infection due to S. heidelberg, S. enteritidis, S. newport, and S. typhi-murium. This general picture is supported by the findings in a number of outbreaks and by the relation between the salmonella types isolated in the investigation of abattoirs and in the population receiving meat from them (19).

Table 6 also indicates that home-produced poultry is a source of S. thompson, S. typhi-murium, and S. saint-paul infection.

Salmonella typhi-murium is spread widely throughout our home-produced domestic animals and poultry as well as in imported human foods, so that an understanding of its sources comes from a knowledge of the phage types found in man and the relation of these to the types found in the possible sources of infection. Miss B. R. Callow, working with Dr. A. Felix, devised an effective phage-typing scheme for S. typhi-murium (20–23) which opened a new avenue of approach. The extension and use of the method by Anderson (24–26) has yielded information about the probable sources of human infection with S. typhi-murium in the United Kingdom in terms of the frequency distribution of phage types in the various host animals. Anderson believed that the types common in man should also be common in the animals responsible

Table 5.–*Salmonella serotypes from domestic animals in England and Wales, 1956-61*

Serotype	Bovines[1]	Chickens[2]	Ducks	Pigs	Sheep	Turkeys
abaetetuba	1	...	...	...	...	...
aberdeen	...	...	...	1	1	...
abortus ovis	...	...	...	...	2	...
adelaide	...	...	...	1	...	...
africana	...	1	...	...	...	...
agama	21	3	...	4	...	...
alachua	...	...	...	2	...	...
amager	...	1	1	...	...	...
anatum	4	5	11	54	...	4
bareilly	1	...	...	1	...	...
binza	1	1	...	1	...	2
blockley	...	1	...	...	...	...
bovis morbificans	...	...	...	14	1	...
braenderup	2	...	...	...	...	1
brancaster	3	...	3	...	...	2
brandenburg	...	...	...	3	...	...
bredeney	3	...	...	11	...	...
bury	1	...	...	...	...	...
butantan	...	...	...	1	...	...
california	1	1	...	2	1	4
chailey	...	1	...	...	...	...
chester	...	...	...	1	...	...
chicago	...	...	1	...	...	...
cholerae-suis	2	1	...	32	1	...
coleypark (new type)	...	2	...	...	...	...
cubana	1	1	5	4	...	2
derby	1	...	...	7	...	3
dublin	109	4	1	20	5	...
eastbourne	...	...	...	1	...	...
emek	7	...	...	...	...	...
enteritidis	9	3	1	15	...	1
essen	...	...	...	1	...	...
frintrop	...	...	...	1	...	...
gallinarum	...	12	...	...	...	...
give	3	...	2	8	...	...
godesberg	...	2	...	...	...	...
havana	...	...	...	...	1	...
heidelberg	20	...	...	36	5	...
hvittingfoss	...	1	...	...	...	...
infantis	...	...	...	2	...	1
kaapstad	...	...	...	1	...	...
kentucky	...	...	...	1	...	...
kiambu	...	...	...	11	...	...
kinshasa	1	5	...	...	...	8
lexington	...	1	...	1	...	...
liverpool	2	...	...	3	...	...
livingstone	...	5	...	1	...	...
llandoff	1	...	...	...	...	...
london	1	...	...	3	...	...
manchester	...	1	...	...	...	...
manhattan	2	...	...	2	...	...
meleagridis	1	1	...	2	...	...
menston	...	139	...	5	...	1
minneapolis	...	...	...	...	...	1
minnesota	...	...	...	4	...	...
mishmar-haemek	...	...	...	1	...	...
montevideo	...	1	...	3	1	3
muenchen	...	5	...	3	...	7
muenster	...	...	...	6	...	...
new haw	...	...	...	...	...	2
newington	...	...	...	2	...	...
newport	1	1	...	10	...	...
niloese	1	...	...	...	...	...
oranienburg	3	...	...	...	1	...
orion	...	...	...	2	...	1
paratyphi B.	...	...	...	...	...	1
poona	...	...	...	1	...	...
pullorum	...	21	...	...	1	1
reading	5	...	...	2	...	...
richmond	1	...	...	...	...	...
ruiru	1	...	...	...	...	1
saint-paul	...	21	1	13	...	11
san-diego	...	...	...	1	...	...
schwarzengrund	...	4	...	13	...	1
senftenberg	1	5	...	4	...	8
simsbury	1	...	...	...	...	...
stanley	1	...	...	1	...	1
stanleyville	1	...	...	...	...	2
taksony	...	...	...	2	...	...
taunton	...	...	...	2	...	...
tennessee	5	...	...	1	...	...
thompson	7	230	3	12	2	3
typhi-murium	410	506	306	80	5	115
vejle	5	...	...	...	...	...
virchow	...	...	...	17	...	...
winchester	...	...	...	1	...	...
worthington	...	6	...	2	...	1
Total strains	541	992	335	436	26	188
Total types	38	32	11	57	13	27

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[1] Includes cows, calves, heifers, steers, bullocks, oxen, etc.

[2] Chicks, hens, fowl; includes older hens and cockerels.

Table 6.—*Animal sources in the United Kingdom of 7 common human salmonella serotypes*

[Number of strains 1958-62]

Serotype	Animal species					
	Bovine[1]	Pig	Sheep	Duck	Fowl[2]	Turkey
typhi-murium[3]	410	80	5	306	506	115
heidelberg . .	20	36	5	. . . .	. . . . .	. . . . .
enteritidis . .	9	15	. . . . .	1	3	1
paratyphi B. .	. . . . . .	. . .	. . . . .	. . . .	. . . . .	1
thompson . . .	7	12	2	3	230	3
saint-paul. . .	. . . . . .	13	. . . . .	1	21	11
newport	1	10	. . . . .	. . . .	1	. . . . .

[1] Includes cows, calves, heifers, steers, bullocks, oxen, etc.
[2] Includes chicks, chickens, hens, and older birds.
[3] Includes Dr. E. S. Anderson's figures.

for human infection, and that these animals should be identifiable, provided that the types concerned showed a tendency to predominate individually in particular animal hosts. Moreover, common human types not found in animals in the United Kingdom would probably be imported from abroad. The results of this work have confirmed these predictions. Briefly, the findings are as follows (Dr. E. S. Anderson, personal communication):

1. There is a wide scattering of phage types in man; in 1961 and 1962 about 80 phage types were encountered. The following observations refer to the 21 most common types, which together constituted 3,939 (85 percent) of the 4,641 human cultures examined.
2. Eight phage types, responsible for 46 percent of human infections in the years 1961 and 1962, were most frequently found in bovines.
3. One phage type, responsible for 18 percent of human infections, was most common in chicks.
4. Two phage types, responsible for 8 percent of human infections, were most common in ducks.
5. One phage type, responsible for 17 percent of human infections, was most common in bovines, but also occurred fairly frequently in chicks, ducks, turkeys, and pigs.
6. One phage type, responsible for 8 percent of human infections, was predominant in bovines, but also common in chicks and turkeys.
7. One phage type, responsible for 8 percent of human infections, was shared fairly evenly between bovines and chicks.
8. Phage types common in man but rare in, or absent from, British livestock were probably imported. For example, one such type has been isolated from Australian beef.
9. Of 1,119 animal cultures of S. typhi-murium examined in 1961 and 1962, only 3 were isolated from sheep, which clearly are unimportant as a source of S. typhi-murium human infection.

Table 7 shows that the 11 types predominating in bovines, chicks, and ducks produced in the United Kingdom were responsible for 72 percent of human infection. It should be remembered that, although a type may show a heavy predominance in a single host, it occurs nevertheless in most other farm animals.

At present, it is not known whether S. typhi-murium phage types which are associated with a particular animal species in the United Kingdom are associated with the same or any other species in other parts of the world.

Table 7.—*Relation of phage types of* S. typhi-murium *to human and animal infection*

Host	Number of predominant phage types	Percentage of human infection due to predominant phage types
Bovines[1]	8	46
Fowl[2]	1	18
Ducks	2	8
Total	11	72

[1] Includes cows, calves, heifers, steers, bullocks, oxen, etc.
[2] Includes chicks, chickens, hens, and older birds.

SALMONELLA IN ANIMAL FEEDING STUFFS

In order to assess the importance of animal feeding stuffs to the problem of animal salmonellosis, it is necessary not only to know the degree of infection of the raw materials, and the serotypes therein, but also the degree of contamination of the prepared foods which are fed to the animals.

In the United Kingdom, large numbers of animals and poultry are bred and reared in confined areas; also, animals and poultry bred in one locality are reared in another. During transportation, the animals or birds are crowded together and are then mixed with other stock. It is known that infected calves from Wales have introduced salmonella into herds in eastern parts of England where they were to be reared. These methods of intensive farming result in conditions which are ideal for cross infection to take place.

Comparatively small amounts of raw animal foods are used in the feeding of young animals and the main foods are processed, being fed as mashes, crumbs or pellets, according to the species and the age of the animal. In some areas, mixtures are used by the farmer who adds his own cereals; the mixtures contain protein meals, minerals, vitamins, etc. The use of such mixtures is declining, but where the complete food is a wet mash and when sufficient time elapses before feeding to animals, then salmonella multiplication can occur. In the United Kingdom, there is increasing use of complete foods which are heated during processing and, therefore, contain very few or no salmonellae.

Reports (27, 28) were published which showed that salmonella-infected raw materials were used in animal feeding stuffs. The most heavily contaminated raw materials were imported meat meals (29) and Indian crushed bones (30). It was also shown that, after the heat treatment of these raw materials, the number of positive samples, the number of salmonellae present in a positive sample, and the number of different salmonella serotypes isolated were greatly reduced. It was also shown that processed foods, such as pellets and crumbs, were almost free from salmonellae.

Table 8 gives the frequency distribution of some important salmonella serotypes isolated from raw materials used in feeding stuffs, complete foods, and domestic animals and associated with human incidents. The number of positive samples in complete processed foods was 126; whereas in the raw materials, it was 789. Although the percentage of positive samples due to S. oranienburg and S. senftenberg was greater in the complete foods, the actual number of isolations was reduced. Many strains of S. senftenberg were heat-resistant and survived processing.

With the exception of S. typhi-murium, the most important types found in animals were almost absent in complete animal foods and were very rare in raw materials. These types were S. cholerae-suis, S. dublin and S. gallinarum-pullorum, which are host-specific to pigs, bovines, and fowl, respectively; also S. enteritidis, S. heidelberg, S. menston, S. saint-paul, S. thompson, and S. typhi-murium, for which there is evidence of animal host association in the United Kingdom. Many of the strains of S. typhi-murium in raw materials were isolated from meat meals produced in the United Kingdom and could have originated from native animals.

S. anatum, S. bredeney, S. meleagridis, and S. senftenberg were common in raw materials and relatively common in complete foods; they were found in domestic animals, particularly pigs, often enough to suggest a direct association with infected food.

S. montevideo, S. newport, S. oranienburg, and S. tennessee were common in food stuffs but were rare in the series of animals studied at this time.

It is difficult to assess the relation of animal feeding stuffs and infection in animals to the large number of other serotypes indicated in Table 8.

Table 8.—*Salmonella isolated from raw materials, complete animal foods, animals, and sources associated with human incidents*[1]

Serotype	Raw materials[2] (1956-61)	Complete Foods[2] (1956-61)	Bovine[2] (1956-61)	Pigs (1958-62)	Poultry[3] (1958-62)	Comments	Isolates associated with infection in humans 1955–62
Total number of strains . .	789	126	2,116	436	1,515		35,876
cholerae-suis	. . . .	. . . .	(2)	7	(1)	Almost absent in animal foods. Host-specific and host-associated types.	(101)
dublin	(1)	. . . .	65	5	(5)		(214)
enteritidis	(1)	. . . .	1	3	(5)		3
gallinarum	. . . .	. . . .	. . . . . .	. . . . . .	[4]2		. . . .
pullorum							(6)
heidelberg	(2)	. . . .	2	8	. . . .		4
menston	(3)	. . . .	. . . . . .	1	9		(107)
saint-paul	(3)	. . . .	(7)	3	2		1
thompson	(1)	. . . .	(9)	3	15		2
typhi-murium	4	2	26	18	61		68
anatum	6	3	(5)	12	1	Evidence of animal food and species association.	1
bredeney	3	2	(4)	3	. . . .		1
meleagridis	4	2	. . . . . .	(2)	(1)		(122)
senftenberg	9	19	(2)	1	1		(70)
montevideo	7	(1)	. . . . . .	1	(4)	Common in animal food, rare in animals.	(111)
newport	2	. . . .	. . . . . .	2	(1)		2
oranienburg	3	9	. . . . . .	. . . . . .	. . . .		(82)
tennessee	2	2	(6)	(1)	. . . .		(52)
Other serotypes	58	60	5	33	7		15

[1] Percentage of strains isolated. Numbers in parentheses indicate numbers of isolations which were too small to include as a percentage.
[2] Figures supplied by Dr. J. H. McCoy. Includes cows, calves, heifers, steers, bullocks, oxen, etc.
[3] Includes chicks, chickens, hens, older fowl, turkeys, and ducks.
[4] Unrealistic number as such types are rarely sent to the Salmonella Reference Laboratory.

DISCUSSION

Salmonellosis is fairly common in the United Kingdom. Because of the National Health Service, it is probable that the notification of human salmonellosis is almost complete for the outbreaks, and a fair proportion of family outbreaks with a smaller proportion of sporadic cases are also recorded. With this amount of recording, it is probable that information on the importance of the various serotypes is reasonably accurate.

I have emphasized the fact that 7 serotypes are responsible for more than 80 percent of human infection and that these types were among the 10 most common in each year for 5 years from 1958 to 1962. The main problem is to determine the source of these types, and we now believe that the major source of these strains is our own home-produced domestic animals and poultry. This source is shown by the close correlation of the serotypes found in our domestic animals and those in man; in addition, Dr. E. S. Anderson's results on the phage types of S. typhi-murium, our most important serotype, confirm that the major source of this infection is home-produced bovines and poultry.

I have alluded briefly to farm practice in the United Kingdom, as it is thought that this contributes to the picture of animal salmonellosis and so to human infection. The concentration of animals and birds which results from modern methods of intensive production is ideal for cross infection to occur. We believe that such methods have resulted in producing host-associated typed. Dr. E. S. Anderson's results suggest that 8 phage types of S. typhi-murium are associated with bovine animals, and that certain phage types are associated with chicks and with poultry. My results support Dr. Anderson's views and, in addition, suggest that S. thompson, S. menston, and S. saint-paul are associated with poultry; in fact, we have evidence that S. thompson, S. menston, and S. typhi-murium are egg-transmitted in some large breeding flocks which produce a considerable proportion of the poultry in the United Kingdom.

The association of pigs and bovines with S. heidelberg, S. enteritidis, and S. newport is also very obvious. The sheep is rarely a source of salmonellosis, a fact which is true of every country for which reports are available.

The pig is different from other domestic animals in that a large variety of different serotypes are isolated from this species. The association of salmonellae in pig foods and the symptomless excretion of the same types by pigs fed on the food is well established (31, 32).

There is not, as yet, enough information about the carrier rate among animals to be dogmatic on the subject of host-association, but it is believed that certain phage types of S. typhi-murium and possibly S. heidelberg can be regarded as bovine-associated types.

The factors which have led to this host association in the United Kingdom may not operate in other countries; and, in any case, there is no reason to believe that serotypes which are host-associated in the United Kingdom will necessarily be associated with the same or any host in other countries. It is unwise to make guesses; but, from published figures, I wonder whether S. oranienburg and S. montevideo are chicken-associated in the United States and whether S. bareilly is chicken-associated in the Netherlands.

Animal feeding stuffs in the United Kingdom — and I would stress United Kingdom — probably do not play a large part in human infection. The type of feeding stuffs and the method of use operate to ensure a minimal chance of infection. Nevertheless, when these products are fed to young animals, which are particularly susceptible, some infections develop. Because the rate is small, it must not be disregarded. It is possible that serotypes which are at present unimportant may become animal host-associated and so contribute to human infection as has happened with S. menston. This serotype was isolated from 28 human incidents between 1949 and 1959; but, after it became egg-transmitted, it was isolated from 103 incidents between 1960 and 1962.

So far discussion has centered round the few serotypes which cause more than 80 percent of human infections. The remaining human infections are due to some 70 to 80 serotypes. A few of these types, S. dublin, S. cholerae-suis, and S. bovis-morbificans, are commonly found in our own domestic animals. The United Kingdom imports large amounts of human foods, some of which are infected with salmonella; such foods have been the source of many human outbreaks of S. paratyphi B and some outbreaks due to other serotypes. Certain serotypes are found consistently in imported foods, for example S. bareilly and S. meleagridis in coconut. For many human infections, however, particularly those occurring only once or twice in a period of years or occurring more frequently in but one particular year, it is impossible to trace the source, whether from native stock or from imported foods. These imports must be considered as possible sources of salmonellosis, particularly of the 15 percent due to other serotypes (Table 8), as well as the named serotypes; also, Dr. Anderson has evidence that certain phage types of S. typhi-murium infection in humans were introduced in imported food.

I have discussed the general picture of salmonellosis in the United Kingdom without mentioning hospital outbreaks, which are responsible for the majority of institutional outbreaks of all kinds. The degree of cross-infection in hospitals in the United Kingdom was analyzed by Taylor (33) who attempted to assess various factors. The principles underlying hospital infection are basically the same as those causing animal infection. These are:

1. The herding together of those susceptible to infection;

2. The presence of an infected individual of the same animal species who has no control over the voiding of feces-individuals such as the baby, the adult with diarrhea, or animals;
3. Less important as an over-all cause in hospitals and in animals, the infected food.

I do not intend speaking on the control of salmonellosis for this is a vast subject involving the way of life of ourselves and our domestic animals.

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Salmonellosis in Canada

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The purpose of this paper is to outline the problem of salmonellosis in Canada as seen by an epidemiologist and a bacteriologist. As a functional definition we consider "salmonellosis" to include the presence of salmonellae in foods as well as in humans and animals, including poultry. With respect to Canada, the paper outlines the reported incidence of typhoid and paratyphoid fever and of other human salmonella infections, salmonellae in animals and processed human foods, serotypes identified in Canada with commentary, preliminary data for 1963, and conclusions.

INCIDENCE OF TYPHOID AND PARATYPHOID FEVER

In 1924, when typhoid and paratyphoid combined became reportable on a national basis, 2,199 cases were recorded. The annual incidence dropped gradually to 276 in 1962. The corresponding rates per 100,000 population are 24.3 and 1.5, respectively. Deaths from these diseases decreased from 603 in 1924 to only 3 in 1962. Supplementary reports from provincial epidemiologists show that, in the 5-year period 1958-1962, of the reported cases of typhoid and paratyphoid A and B, the proportion was respectively 45.4 percent, 2.3 percent, and 52.3 percent. These reports also show that on December 31, 1962, the following number of carriers were under active surveillance: typhoid — 174; paratyphoid A — 2; paratyphoid B — 48; and typhoid and paratyphoid combined — 25.

Although the incidence of typhoid and paratyphoid fevers in Canada has dropped to a low level, dramatic evidence of the need for continuous sanitary control was the outbreak of milk-borne typhoid in Montmagny, Quebec, in March 1959 (1). Of 197 cases reported, 188 occurred in a population of 6,000. The source of the epidemic was raw milk from one dairy. The well of this dairy had dried up, and untreated river water was used for cleaning the dairy equipment. Eight of the 10 persons in the dairyman's family developed typhoid fever.

INCIDENCE OF OTHER HUMAN SALMONELLA INFECTIONS

On the other hand since 1955, there has been a steady and marked increase in the reported incidence of human salmonella infections, other than typhoid and paratyphoid; this increase has been attracting more and more the attention of our control officers. The need for this increased surveillance was emphasized by the recent report in Alberta of a hospital outbreak of salmonella food poisoning (2). In the first week of August 1962, an explosive outbreak of diarrhea and vomiting occurred among the staff and patients of a large hospital. A total of 116 cases were reported. More cases were suspected, but they were not examined or reported. Of 74 bacteriological confirmations, 59 were positive for *Salmonella thompson*, 10 for *S. heidelberg*, and 5 for both organisms. The source of this outbreak was traced by a public health inspector to meringue powder which had been used in the hospital kitchen to prepare topping for custard. A count of organisms in the first specimen of the meringue powder examined showed a uniform distribution of approximately 7 million salmonellae per gram of powder.

Human salmonella infections other than typhoid and paratyphoid are not notifiable on a national basis in Canada unless they occur as food poisoning outbreaks or as epidemic diarrhea of the newborn. Consequently, for information on other human salmonella infections and non-human salmonella sources, reliance must be placed on laboratory reports. Provincial Public Health Laboratories submit monthly reports of thier total salmonella, shigella, and pathogenic *E. coli* isolations to the National Enteric Reference Center at the Laboratory of Hygiene, Department of National Health and Welfare. This center identifies referred specimens serologically and prepares reports.

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Table 1.–*Salmonellae in man and animals, Canada 1953-62*[1]

Serotype	Source[2]	1953	1954	1955	1956	1957	1958	1959	1960	1961	1962	Total
anatum	H	1	2	1	24	6		2	4	4	13	57
	A	1	2	6	4	3	1	1	6			24
bareilly	H	15	35	22	8	9	13	31	13	7	7	160
	A	5	74	10	6	29	6	3	6	15	2	156
blockley	H				1		1	30	23	38	23	116
	A							5	2	23	3	33
brandenburg	H	2	2		3	4	6	11	2	9	6	45
	A										1	1
bredeney	H	5	7		1	5	3		5	4	18	48
	A	12	8	12	12	8	5	3	12	14	11	97
canada	H								59	3	1	63
	A											–
cholerae-suis	H	3	1		1		1	5		2	17	30
	A	4	8	8	20	20	9	19	32	43	49	212
derby	H	3	5	7	1	4	39	27	7	13	2	108
	A	6	7	5		6	2	2	2	5	6	41
enteritidis	H	5	3	3	5	5	7	26	23	53	34	164
	A	5	2	2	6	2	19	4	1	11	1	53
heidelberg	H	24	47	58	174	183	143	258	280	384	550	2,101
	A	1	10	3	3	15	29	101	60	115	86	423
infantis	H		2		2	23	15	7	51	32	35	167
	A					1	1	4	–	6	45	57
kentucky	H	6	2	1	1	2	6	8	1	1	2	30
	A		9	1	2		4	2	5	1	6	30
manhattan	H	2		1	2	1	6	19	52	9	2	94
	A		1		4			2		1		8
montevideo	H	6	12	15	149	64	125	64	72	21	28	556
	A		3	3	6	1	3	2	19	21	28	86
muenchen	H		5	3	11	3	1	10	9	5	16	63
	A				1			1			1	3
newington	H		2	2	1	1	2		3	6	6	23
	A	1	3	2	10	3	6	6	32	34	14	111
newport	H	21	31	56	50	100	160	115	100	77	206	916
	A	5	1		1	1	5	10	3	6	29	61
oranienburg	H	19	32	9	24	15	21	13	13	29	35	210
	A		1	2	2	2	1	5	17	11	3	44
panama	H				4	2	3	9	3	5	9	35
	A									2		2
paratyphi A	H	3	2	3	1	5	16	1		6	1	38
	A											–
paratyphi B	H	107	119	88	116	78	82	86	92	135	106	1,009
	A							1		3	7	11
saint-paul	H				4	2	3	1	3	12	27	52
	A					1	1		1	3	3	9
san-diego	H		1	2		5	14	16	11	11	9	69
	A	1						6	22	13	3	45
senftenberg	H		2		3	3	1		1	2	3	15
	A			1	3		3		9	5	19	40
tennessee	H	22	38	7	28	26	58	37	36	21	52	325
	A	2	1	2	3	2		5	5		11	31
thompson	H	15	31	23	56	65	32	18	256	554	690	1,740
	A	25	69	16	58	34	13	21	9	7	23	275
typhi	H	213	312	126	241	196	141	151	145	126	128	1,779
	A											–
typhi-murium	H	194	384	304	290	401	497	483	423	435	395	3,806
	A	54	88	56	81	111	64	90	83	105	108	840
worthington	H		1	7					1	2		11
	A					1			12	18	33	64
Other types	H	7	6	7	23	12	28	20	34	48	38	223
	A	1	8	1	3	2	1	8	13	10	42	89
Unidentified	H	2		3	2	2	2	10	8	8	3	40
	A						4	1	4	6		15
Totals	H	675	1,084	748	1,226	1,222	1,426	1,458	1,730	2,062	2,462	14,093
	A	123	295	130	225	242	177	302	355	478	534	2,861

[1] Source: National Enteric Reference Center

[2] H–Human; A–Animal and poultry.

Table 1 is based on data from the foregoing sources. It shows the more common salmonella serotypes isolated in Canada from man and animals in the 10 years, 1953 to 1962. While these data are limited to isolations in public health laboratories, they do indicate trends. The isolations from humans do not include repeat or duplicate identifications. Even if we assume a greater awareness of the problem on the part of the medical profession, better diagnoses, more accurate reporting year by year, these factors are probably not sufficient to explain the increase from 675 cases in 1953 to 2,462 cases in 1962.

In the 10 years, 1953 to 1962, the most frequent serotypes isolated from man other than S. typhi and S. paratyphi A and B have been S. typhi-murium, S. heidelberg, and S. thompson. Several of the other strains commonly found in man were S. newport, S. montevideo, and S. tennessee.

Other serotypes which were also found regularly, though in smaller numbers, were S. oranienburg, S. enteritidis, S. bareilly, and S. derby and, during the last half of the 1950's, S. infantis and S. blockley. Examples of salmonella types which have been found only once are S. adelaide, S. canoga, S. chailey, S. coeln, S. vancouver, and S. denver.

INCIDENCE OF SALMONELLA INFECTIONS IN ANIMALS

The data for animals in Table 1 concern isolates mainly from sick or dead animals or poultry. Among the animal population, S. typhi-murium and S. heidelberg have consistently been the types most frequently and regularly identified. In the period 1953 to 1957, S. thompson was also a common strain in animals, particularly in poultry, but isolations of this type from animal sources have steadily declined in recent years. It is certainly noteworthy that the most frequently found salmonella types in disease in man are the same types most commonly isolated from animals. Is this purely coincidence? Surely this is an argument in favor of the direct relationship of the disease in animals to that in man. Several of the other more commonly found animal strains are S. cholerae-suis, S. bareilly, S. newington, and S. montevideo; but these — with the possible exception of the last strain — like S. gallinarum-pullorum, would appear to be more parasitic for animals than for man.

SALMONELLAE IN PROCESSED HUMAN FOODS

The rapid and marked increase in human infections with S. thompson and S. heidelberg in recent years is probably related to their presence in very large numbers in foods which have been prepared for widespread use in Canadian homes. Study of a large number of human foods on the market, particularly egg-containing processed foods, has revealed many salmonella types. The most common serotypes found in the past 4 years are shown in Table 2. Of these the most frequent isolations are S. thompson, S. tennessee, and S. heidelberg. Not only have human foods probably played a major role in dissemination of the disease to man, but the finding of large numbers of salmonellae in many prepared animal foods and in rendering plants probably explains much of the spread of these organisms among the animal population.

It should be pointed out, however, that the food products found to contain salmonellae by the Food and Drug Laboratories of the Department of National Health and Welfare represent units or lots which are prevented from reaching the market or consumer.

Table 2.–*Salmonellae in processed human foods*[1] *Canada, 1959-62*[2]

Serotype	1959	1960	1961	1962
bredeney	. . . .	. . . .	. . . .	11
gallinarum-pullorum	48	5	. . . .	16
heidelberg	. . . .	7	7	87
infantis	. . . .	7	6	13
manhattan	. . . .	5	. . . .	7
montevideo	. . . .	19	3	18
oranienburg	. . . .	23	5	11
senftenberg	. . . .	2	. . . .	13
tennessee	3	11	1	115
thompson	1	79	39	458
typhi-murium.	3	2	1	9
worthington	. . . .	4	. . . .	26
Other	2	19	2	53
Total	57	183	64	837

[1] Primarily cake mixes and various egg products.

[2] Source: National Enteric Reference Center

At the present time, processed food products containing salmonellae must be retained by the manufacturer until retreatment and re-examination show these products to be free from salmonellae. Many isolates are also the result of biased selection; depending on the previous record of a processing plant for producing salmonella free or salmonella contaminated products.

SALMONELLA SEROTYPES IDENTIFIED IN CANADA

There has been a total of 101 serotypes reported in Canada, up to the end of 1962. It is particularly indicative of the problem of salmonellosis in Canada that in the last decade 1953-1962, 45 or almost half of all types reported in Canada were reported for the first time. Most of these have been isolated from both man and animals, but some have so far been isolated only from man, and others only from non-human sources. Twenty-six of the 101 serotypes have been isolated only once. Many types produce only a very small number of infections year after year, but never entirely disappear. There are only comparatively few that have become well established in either man or animals or both and continue every year to be the principal contributors to salmonellosis in Canada. This is perhaps one of the most intriguing problems in this whole field. Why does one type appear once and like a comet disappear, and another type, once it has made an appearance, continue to multiply and spread?

S. heidelberg presents an excellent example of the type that once it has established a beachhead continues to spread and becomes a major part of the salmonella picture. This type made its first appearance in Alberta in 1952, where it caused considerable loss in 3 flocks of turkey poults and one of baby chicks. All these baby birds had originated in one hatchery is southern Alberta. The turkey eggs had been imported from Oregon in the United States and hatched poorly. During the remainder of the year, four human infections due to S. heidelberg occurred, three of them in rural areas near the infected hatchery. From these small beginnings, this serotype spread fairly rapidly across Canada in both the human and animal populations. From being unknown in 1951, it rose to the third most frequently found type in human infections in 1956 and has continued as one of the three most common serotypes. Coincidental with this increase in human cases, a similar increase of this serotype occurred in animal isolates, particularly poultry. By 1958, it was the second most commonly found type in animals; and, in 1959 and 1961, S. heidelberg was the most commonly found type from this source. While the evidence is lacking to demonstrate the exact means whereby this organism was spread across the country, there is little doubt that poultry has played a significant role in this spread. Recently, the isolation of this type from animal feeds, cake mixes, and egg products suggests other means of distribution in both the human and animal population. During the past 10 years, this type has been involved in many food poisoning outbreaks in which cooked turkey, particularly, and turked dressing as well as other foods have been incriminated.

On the other hand, S. canada is an example of the type which appears suddenly then disappears. As the name suggests this was a new Canadian serotype, described by Yurack et al. (3). It was isolated simultaneously in two widely separate areas of the country. From February to August 1960, this organism spread through 8 of the 10 provinces with a total of 59 isolations from cases and contacts. As with most human salmonella infections, the elderly and especially the very young were particularly susceptible; 36 of the 59 infections were in children under 5 years of age. The children often remained carriers for a considerable time. The widespread occurrence and rapid dissemination of this organism are highly suggestive of some packaged food being the vehicle, but attempts to discover such a source failed.

Since this period, S. canada has practically disappeared from the Canadian scene. In 1961, four isolations were reported in Ontario, but all were in the same family; a 10-week-old infant, both parents, and another child, who was one of the original cases in the previous year, were all carrying the organism. In 1962, one isolate was reported, also in Ontario, and it represents the only new episode since the original outbreak in 1960. No further isolations of this type have been reported to date.

S. thompson is another interesting serotype whose incidence in humans suddenly skyrocketed in 1960; by 1962, it was the most frequently found organism in human infections. The history of this type and its implications is the subject of a separate presentation at this conference by Dr. F. S. Thatcher of the Food and Drug Laboratories, Department of National Health and Welfare.

HUMAN SALMONELLOSIS IN 1963

Although data for 1963 are as yet incomplete, certain trends and observations are noteworthy. As mentioned previously, in 1962, S. thompson was the serotype most frequently isolated from man in Canada. However, in 1963, S. typhi-murium accounted for between 30 and 40 percent of all human infections and is followed by S. thompson, S. heidelberg and S. newport. The total incidence of human salmonellosis has increased over that of 1962; this was due primarily to increased infections in 2 provinces, Ontario and Quebec. In both provinces, this increase was due mainly to S. typhi-murium, while in Ontario a large outbreak due to S. thompson was also a major factor.

It was in June that a marked increased in the incidence of S. typhi-murium was noted in the two neighboring provinces mentioned above; this increase continued to the end of the year. The Laboratories Division, Ontario Department of Health, reported 588 isolates of S. typhi-murium from June to September 1963. Many, if not most of these, are suspected to be due to the consumption of meringue which had been in use in one bakery which supplied pies to the restaurant trade over a wide area. In the same province at the end of August, another outbreak due to S. typhi-murium occurred at a wedding reception attended by some 600 persons. One hundred cases were positive bacteriologically, and S. typhi-murium was isolated from "cheese pastry," one of the foods served at the reception. Further, in Ontario, the increase in S. thompson isolations mentioned previously was due to a large institutional outbreak early in the year which, over a period of time, involved 475 cases predominately caused by S. thompson. The source of this outbreak was reported to have been meringue powder purchased in large quantity.

With respect to salmonellosis in foods and animals during 1963, egg products, including meringue powders, continued to be a prime source of salmonellae in Canada. S. thompson was still the salmonella

Table 3.—*Salmonella types most frequently identified, by source, Canada, 1953-62*[1]

Man			Animal			Food[2]		
Serotype	Number	Percent	Serotype	Number	Percent	Serotype	Number	Percent
typhi-murium	3,806	27.0	*typhi-murium*	840	29.4	*thompson*	577	50.4
heidelberg	2,101	14.9	*heidelberg*	423	14.8	*tennessee*	130	11.3
typhi	1,779	12.6	*thompson*	275	9.6	*heidelberg*	101	8.8
thompson	1,740	12.3	*cholerae-suis*	212	7.4	*gallinarum pullorum*	69	6.0
paratyphi B	1,009	7.2	*bareilly*	156	5.4	*oranienburg*	41	3.6
newport	916	6.5	*newington*	111	3.9	*montevideo*	40	3.5
montevideo	556	3.9	*bredeney*	97	3.4	.....	.....	.....
tennessee	325	2.3	.....	.....	.....	.....	.....	.....
Other salmonellae	1,861	13.3	Other salmonellae	747	26.1	Other salmonellae	187	16.4
Total	14,093	100.0	Total	2,861	100.0	Total	1,145	100.0

[1]Source: National Enteric Reference Center

[2]Almost no isolations were made prior to 1959.

type most frequently isolated from these sources, followed by *S. tennessee.* It is also significant that in 1963, *S. typhi-murium* was the third most commonly found salmonella from cake mixes and egg products. As might be expected, *S. typhi-murium* and *S. heidelberg* continue to be the most common types in animals and poultry.

RELATIONSHIP OF SALMONELLA IN MAN, ANIMALS, AND FOODS

Table 3 shows the relationship between the predominant types of salmonellae found in man, animals, and food. The most frequently identified serotypes in man are also those most commonly isolated from animals. *S. thompson* and *S. heidelberg,* two of the major contributors to salmonella disease in man and animals, have also recently been found extensively in foods. It is surely more than coincidental that the incidence of *S. thompson* in human infections increased sharply at the same time this serotype was isolated in large numbers from a variety of food products.

Table 4.—*Salmonella types most frequently identified*
[Canada and the United States]

Rank	Canada 1962[1]		United States 1963[2]	
	Man	Animal	Man	Animal
1	*thompson*	*typhi-murium*	*typhi-murium*	*typhi-murium*
2	*heidelberg*	*heidelberg*	*derby*	*heidelberg*
3	*typhi-murium*	*cholerae-suis*	*heidelberg*	*infantis*
4	*newport*	*infantis*	*newport*	*anatum*
5	*typhi*	*worthington*	*infantis*	*montevideo*
6	*paratyphi* B	*newport*	*enteritidis*	*newport*

[1]Source: National Enteric Reference Center.
[2]Source: CDC Salmonella Surveillance Report No. 21, Feb. 3, 1964.

COMPARISON OF SALMONELLAE IN CANADA AND THE UNITED STATES

Table 4 compares the salmonella types most commonly found in man and animals in Canada during 1962, with the most frequently found types from the same sources in the United States during 1963. It is particularly noteworthy that *S. typhi-murium* and *S. heidelberg* are predominant types in man and animals in both countries. It is also evident that there are differences in frequency of isolation of the other salmonella types which are major contributors to salmonellosis in man and animals in both countries. An outstanding example is the high incidence of *S. thompson* in Canada in man as compared to the relatively low incidence in the United States.

CONCLUSION

(1) Human salmonellosis in Canada is steadily increasing; greater numbers of salmonellae are being isolated from animals and a wide variety of processed human foods.

(2) While the case and the symptomless carrier are the primary sources of those salmonellae which are specific for man (viz., *S. typhi, S. paratyphi A,* and *S. paratyphi B*), animals, particularly poultry, appear to be the principal reservoir of most of the other salmonella types responsible for infections in man (viz., *S. typhi-murium, S. heidelberg,* and *S. thompson*).

(3) Salmonellosis control can be effectively achieved only by the concerted efforts of persons concerned with human health, animal health, and food processing.

ACKNOWLEDGEMENTS

The authors gratefully acknowledge the cooperation of Provincial Public Health Laboratory Directors and others for submitting cultures and data, which are here reported. The authors are also indebted to Dr. E. T. Bynoe, Director, Laboratory of Hygiene for reviewing the manuscript.

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The Results of Typing Salmonella from Animal Sources in the United States

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The serotyping of salmonella isolates from animal sources was initiated by the Animal Disease Eradication (ADE) Division of the Agricultural Research Service in response to a demand expressed for such a service by directors of publicly and privately supported animal disease diagnostic laboratories. The Division felt that it should, in all fairness, lift part of the burden from the Communicable Disease Center (CDC) personnel who had, up to that time, been providing the only typing service available to animal disease workers except for certain State laboratories. Arrangements were made with CDC for the ADE Division to furnish personnel to assist with the typing in CDC facilities and furnish certain other financial support. After completing these arrangements, this service was offered to State and commercial animal disease diagnostic laboratories. Since 1962, we have been able to continue to provide this service at the National Animal Disease Laboratory in Ames, Iowa. The serotyping of salmonella cultures from animal sources is conducted as part of the Diagnostic Services program of the ADE Division of ARS at NADL. This serotyping of the salmonella is, therefore, a continuation of the work that had been underway for approximately 5 years at the CDC in Chamblee and Atlanta, Georgia, before it was inaugurated at NADL.

At CDC and NADL, 13,457 isolates have been serotyped (Table 1). Of the 10,682 isolates made directly from animals, 10,510 or almost 98 percent were from the principle farm animal species—namely, turkeys, chickens, ducks, geese, swine, sheep, goats, cattle, and horses. However, isolates were submitted from 32 other animal species and 24 other species of birds. There were 86 different serotypes identified from the isolates submitted to us from the 9 farm animal species mentioned above (Tables 2, 3, 4). There were other serotypes that were recovered from one or more of the other species. Of the 86 serotypes from farm animals, there were 15 that were identified only 1 time from these species during the 7 years of typing. There were 7 more serotypes isolated from only 1 species and 17 more isolated from only 2 species. Of the 1,404 isolates identified as *S. pullorum*, only 17 were from turkeys, 2 from cattle, and 1,385 from chickens. *S. gallinarum* was identified 437 times in all—398 times from chickens and 39 times from turkeys. *S. dublin*, on the other hand, was identified 62 times; and 52 times the isolates were from cattle, twice from horses, once from sheep, and only 7 times from chickens. All isolates came from west of the Mississippi River. *S. cholerae-suis* var. kunzendorf continues to be the most common salmonella serotype in swine; in fact, it makes up 60 percent of the isolates from swine. This serotype is largely confined to this species. H_2S negative strains of *S. cholerae-suis* are very rare. *S. abortus-equi*, once a serious cause of disease in bands of mares seems to have all but disappeared from the United States, judging from the cultures submitted to our laboratory. On the other hand, *S. typhi-murium* is a good example of a serotype that is recovered commonly from all species: e.g., 906 from chickens, 685 from turkeys, 27 from ducks, 25 from geese, 123 from swine, 16 from sheep and goats, 444 from cattle, and 104 from horses. In previous publications (1, 2), the prevalence in turkeys was reported to be much greater than in chickens.

At least two factors have contributed to this change. *S. typhi-murium* infection has apparently increased in chicken flocks and has become a problem; whereas formerly it was not. Since the development of an antigen for turkey testing, *S. typhi-murium* infection has been reduced in turkeys.

Table 1.—*Isolates from animal sources submitted for serotyping*

Calendar year	Number typed	Animals	Feed and food
1957	850	786	64
1958	1,733	1,178	555
1959	2,392	1,044	1,348
1960	1,912	1,640	272
1961	2,501	2,167	334
1962	2,702	2,557	145
1963[1]	1,367	1,310	57
Total	13,457	10,682	2,775

[1] Only up to June 30, 1963.

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Table 2.–*Twenty-three salmonella serotypes that constitute 76 percent of the total*

Serotype[1]	Total number	Percent
anatum	795	6.00
blockley	196	1.48
bredeney	238	1.79
chester	262	1.97
cholerae-suis	714	5.39
derby	492	3.71
enteritidis	363	2.74
gallinarum	442	3.33
heidelberg	514	3.88
infantis	472	3.56
montevideo	323	2.43
muenchen	169	1.27
newport	395	2.98
oranienburg	149	1.12
pullorum	1,417	10.70
saint-paul	406	3.06
san-diego	375	2.83
schwarzengrund	170	1.28
senftenberg	186	1.41
tennessee	213	1.60
thompson	144	1.08
typhi-murium	2,750	20.77
worthington	212	1.60

[1] Only the serotypes that were at least 1% of the total are listed.

Table 3.–*Ten salmonella serotypes that were most frequently identified and the percent of total for each year*

Serotype	1957	1958	1959	1960	1961	1962	1963
typhi-murium	18.0	21.0	16.6	19.0	23.0	22.0	26.8
pullorum	12.7	7.6	8.2	21.6	13.2	7.5	6.3
anatum	4.4	9.8	5.2	8.0	7.6	2.6	3.7
cholerae-suis	9.2	2.2	4.0	6.5	3.7	9.2	4.1
heidelberg	6.6	4.2	3.5	4.8	2.7	3.0	7.8
derby	2.2	6.0	7.4	2.6	1.9	2.8	1.2
infantis	2.8	4.5	3.4	2.8	3.5	3.8	3.4
gallinarum	4.0	1.6	3.2	2.2	4.2	5.0	2.4
saint-paul	1.8	1.2	1.5	1.8	3.6	5.2	6.3
newport	3.9	2.8	4.4	3.8	2.6	1.6	1.2

Table 4.–*Relative ranking by frequency of serotypes identified*

Serotype	1957	1958	1959	1960	1961	1962	1963
typhi-murium	1	1	1	2	1	1	1
pullorum	2	3	2	1	2	3	[1]3
anatum	5	2	4	3	3	9	5
cholerae-suis var. *kunzendorf*	3	8	6	4	5	2	4
heidelberg	4	6	7	5	8	7	2
derby	8	4	3	8	10	8	8
infantis	7	5	8	7	7	6	6
gallinarum	[2]6	9	9	9	4	5	7
saint-paul	9	10	10	10	6	4	[1]3
newport	[2]6	7	5	6	9	10	9

[1] Tie for third [2] Tie for sixth

The total number of different salmonella serotypes was the same in turkeys and chickens, 61, while the number from swine and cattle was practically the same, 47 and 43, respectively. Of the 61 serotypes from turkeys and chickens, 10 were identified only once in turkeys and 11 only once in chickens. Of the 47 serotypes from swine, the number of each was small except for S. typhi-murium and S. cholerae-suis var. kunzendorf. Of the 43 serotypes in cattle, there were sizable numbers of more serotypes: namely, S. anatum, S. duesseldorf, S. enteritidis, S. newport, S. taksony, and S. typhi-murium. The 694 isolates of S. cholerae-suis were all from swine except for 13 from chickens, 6 from cattle, and 1 from a horse.

The volume of isolates being submitted has reached a level where very little, if any, further expansion is possible without the addition of personnel. I believe this will be readily apparent from the fact that last fiscal year (1962-1963) one bacteriologist and one assistant serotyped 3,511 isolates; and this fiscal year (1963-1964) from July 1 until March 1, we have already received 3,393 isolates for serotyping. The number of specimens received from commercial laboratories has recently been determined. For the 18 months prior to January 1, 1964, 16 percent were from commercial sources and were submitted by 14 companies.

Our salmonella serotyping laboratory accepts cultures from animal sources referred by State and commercial laboratories, universities, and agencies of the USDA. We ask that as much of the following information insofar as is available be submitted with each culture. Needless to say some are received with very little of these data.

1. Source— animal or feed.
2. If animal — the species, age, organ from which recovered, size of herd or flock, and percent of mortality in the herd or flock from which isolated.

Specimens received in the laboratory are presumed to be salmonella, and no preliminary biochemical tests are done. Agar slant and broth cultures are inoculated, as is a nutrient agar stab. O-antigens (somatic) are made by removing the growth from the agar slant, suspending it in alcohol, heating at 60° C. for one-half hour, decanting the alcohol, and resuspending the sedimented cells in phenolized saline. The resulting antigen is used to determine the O-group of the culture. The broth culture is diluted with formalinized saline; this is the H-antigen used to determine the flagella antigenic components of the culture. Where phase changes are necessary, the Gard technique is used employing motility medium and serum in small petri dishes. The agar stab culture is sealed with a paraffined cork; and after serotyping is completed, this culture is suitably labelled and held for a year. Biochemical tests are performed as necessary to distinguish salmonella that differ biochemically and are alike serologically, and to identify non-salmonella.

Any analysis that one tries to make from the accumulation of data from work of this nature is likely to be misleading. For example, the report of serotyping of isolates received on a State-by-State basis would indicate a great many more submissions from some States than from others. One must recognize that some States do their own serotyping and very seldom call upon our laboratory for assistance. This is evidenced by the fact that of 41 States from which we received isolates for typing during the past 1-1/2 years, during which this work has been carried on in our laboratory, as many as 777 isolates have been submitted from one State and as few as 1 from another. Also, there were nine States from which none were received. Furthermore, there is no assurance that each salmonella culture isolated in the States using our service is submitted to our laboratory for typing. It is likely that most laboratories do not submit S. pullorum or S. gallinarum for typing because of the ease with which they can be identified. Therefore, we have not depicted the number of specimens that have been received from each State. We believe that such a depiction might be misunderstood and interpreted to reflect the incidence of salmonellosis in these States.

Likewise the preponderance of isolates submitted were recovered from chickens and turkeys (Tables 5, 6). There are no doubt several reasons why this is true. First of all there are about 2.75 billion chicks and 100 million turkeys hatched each year. As a result of the National Poultry Improvement Plan and National Turkey Improvement Plan, over 35 million chickens and approximately 3.5 million turkeys are tested for pullorum disease and fowl typhoid. A smaller number of turkeys are tested with S. typhi-murium antigen. Birds from test positive flocks are autopsied, and the isolation of salmonella is attempted. Many isolations that are made as a result of this program are made only from the intestinal contents and not from the body organs of these apparently healthy birds which would seem to indicate that they are healthy carriers rather than diseased birds. Another practice that may further increase the isolations from chickens is the practice recently begun by some breeders of attempting to recover salmonella from "cull" chicks after hatching. When all of these factors are considered, it is probably not surprising that approximately 80 percent of all isolates submitted for typing are from chickens and turkeys.

Table 5.–*Species from which 86 percent of the cultures from animals were isolated*

Species	1957[1]	1958	1959	1960	1961	1962	1963
Turkeys . .	40.6	39.4	25.8	27.9	26.8	30.0	31.6
Chickens .	24.0	19.8	24.6	38.9	38.0	36.6	40.0
Cattle . . .	.24	2.6	6.4	10.6	5.6	6.8	7.4
Swine . . .	10.0	2.4	6.0	7.0	6.9	12.6	15.6
Horses . .	.24	.11	6.3	.8	1.2	2.2	2.4

[1] 1957 poultry unknown 11.84%.

In reporting the results of serotyping of isolates from animal sources we have reported the cultures as submitted from two main categories — animals and food or animal feed. Of course, there is no way by which we can determine that all organisms submitted to our laboratory from the latter category are actually of animal origin. The epidemiologic investigations surrounding the isolations are rarely reported, and therefore there is no way of determining what the original sources of the organism may have been. No doubt strict sanitation in the establishments where animal feeds are processed will reduce the contamination of these feeds with salmonella organisms. As will be reported later by Dr. M. R. Clarkson who will discuss the U. S. Department of Agriculture's salmonella activities, operational sanitary guidelines have been developed in cooperation with the rendering and allied industries. It is expected that they will reduce the likelihood of animal feeds becoming contaminated; and the number of isolates submitted to our laboratory from this source may, in the future, reflect the increased attention that has been given to sanitation by these industries.

Through the use of charts and graphs, we have attempted to depict what we believe are the most significant data that have been accumulated as a result of the serotyping at CDC and NADL.

In summary, these charts of the data accumulated as a result of the serotyping of isolates from animal sources, seem to be the most significant from the over-all point of view. There is almost no end to the interesting information that is recorded in the data. It should always be remembered that the specimens submitted to the laboratory for serotyping probably do not include all isolations made in the States submitting isolates to our laboratory. Additionally, they are not a representative sample of all isolations.

The statement made by one of the authors (1), when reviewing results of serotyping up to 1961, is still applicable. "The material presented perhaps reflects in a general way the salmonella situation in animals in the United States, but it will not be wise to draw too sweeping conclusions from it. Although the numbers of cultures received for serotyping show a general upward trend each year, it is not possible for a worker in a reference laboratory to say whether this is due to an increase in isolations, to better recognition of salmonella species, or to an increasing tendency to take advantage of the serotyping service available."

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Table 6.–*All salmonella serotypes identified from principle animal species*

Serotype	Turkeys	Chickens	Ducks	Geese	Swine	Sheep and Goats	Cattle	Horses	Total
abortus-equi	......	......	......	......	......	......	......	2	2
alachua	14	6	......	......	......	......	......	......	20
albany	6	......	......	......	......	......	......	......	6
amager	5	13	......	......	2	......	......	......	20
anatum	257	83	2	......	18	1	37	12	410
bareilly	8	14	......	......	3	......	3	4	32
berta	9	13	......	......	......	......	......	......	22
binza	30	9	......	......	7	......	......	......	46
blockley	34	148	......	......	5	......	......	......	187
bovis-morbificans	2	......	......	......	1	2	1	......	6
braenderup	8	11	......	......	......	......	......	......	19
bredeney	142	18	2	......	24	......	3	......	189
california	35	15	......	......	3	1	5	......	59
cambridge	5	......	......	......	......	......	......	......	5
cerro	......	6	......	......	......	......	......	......	6
chester	214	91	1	......	1	......	1	......	308
cholerae-suis var. kunzendorf.	......	13	......	......	674	......	6	1	694
corvallis	......	......	......	......	......	......	1	......	1
cubana	23	5	......	......	2	......	1	......	31
denver	1	1	......	......	......	......	......	......	2
derby	70	19	3	......	46	2	4	2	146
dublin	......	7	......	......	......	1	52	2	62
duesseldorf	2	......	......	......	......	......	......	......	2
enteritidis	110	49	......	......	3	22	59	102	345
florida	......	......	......	......	......	1	......	......	1
gallinarum	39	398	......	......	......	......	......	......	437
gaminara	1	......	......	......	......	1	......	......	2
give	52	45	......	......	6	1	......	......	104
habana	......	......	......	......	......	......	1	1	2
hamilton	1	......	......	......	......	......	......	......	1
hartford	......	......	......	......	......	1	2	......	3
heidelberg	299	239	......	......	9	1	3	2	553
illinois	......	3	......	......	......	......	......	......	3
indiana	28	20	1	......	1	......	......	......	50
infantis	106	202	......	......	14	1	4	4	331
java	7	2	......	......	2	......	3	......	14
javiana	9	3	......	......	......	......	......	......	12
johnnesburg	1	1	......	......	......	......	......	......	2
kentucky	24	15	......	......	2	......	2	2	45
lexington	9	3	......	......	1	......	......	......	13
litchfield	11	5	......	......	1	......	1	2	20
livingstone	6	2	......	......	......	......	......	......	8
lamita	......	......	......	......	1	......	......	......	1

Table 6.–*All salmonella serotypes identified from principle animal species*–Continued

Serotype	Turkeys	Chickens	Ducks	Geese	Swine	Sheep and Goats	Cattle	Horses	Total
luciana	......	......	......	......	......	......	1	......	1
madelia	3	1	......	......	......	......	2	7	13
manchester	......	1	......	......	......	......	......	......	1
manhattan	20	4	......	......	4	......	......	......	28
manila	1	......	......	......	......	......	......	......	1
meleagridis	53	19	1	2	5	......	3	......	83
miami	......	......	......	......	5	......	7	......	12
minnesota	4	6	......	......	1	......	2	......	13
mississippi	......	......	......	......	1	......	......	......	1
montevideo	29	125	......	......	16	......	2	10	182
muenchen	122	25	......	......	4	2	3	......	156
muenster	......	1	......	......	......	......	9	2	12
new brunswick	1	......	......	......	......	......	......	......	1
new haw	......	2	......	......	......	......	......	......	2
newington	32	15	......	......	6	......	4	......	57
newport	209	10	1	......	6	3	82	16	327
norwich	......	1	......	......	......	......	1	......	2
nottingham	......	......	......	......	1	......	......	......	1
oranienburg	9	51	......	......	5	......	7	......	72
orion	22	7	......	......	......	......	......	......	29
ohio	......	1	......	......	1	......	......	......	2
panama	17	......	......	......	3	......	......	......	20
pensacola	......	2	......	......	1	......	3	......	6
poona	1	......	......	......	......	......	......	......	1
pullorum	17	1,385	......	......	......	......	2	......	1,404
reading	46	24	......	......	......	......	......	......	70
rubislaw	......	1	......	......	......	......	......	......	1
st-paul	336	67	......	......	6	......	5	5	419
san-diego	339	49	......	......	1	......	4	......	393
schwarzengrund	81	41	......	......	6	......	2	......	130
senftenberg	31	12	......	......	2	......	1	......	46
simsbury	2	......	......	......	3	1	......	......	6
stanley	1	......	......	......	......	......	......	......	1
taksony	1	......	......	......	......	1	26	......	28
tallahassee	......	1	......	......	......	......	2	......	3
tennessee	64	62	......	......	1	......	1	1	129
thomasville	......	1	......	......	1	......	......	......	2
thompson	31	100	......	......	1	......	2	2	136
typhi-murium	906	685	27	25	123	16	444	104	2,330
typhi-suis	......	......	......	......	5	......	......	......	5
uno	1	......	......	......	......	......	......	......	1
urbana	......	1	......	......	......	......	......	......	1
worthington	22	120	......	......	13	......	4	1	160
Total	3,969	4,279	33	27	1,047	58	808	284	10,510

POPULATION AT RISK

MODERATOR : ***Dr. Henry Bauer***

Epidemiology of Salmonellosis

Kenneth W. Newell, M.D.[1]

If human salmonellosis is judged by the suspected amount of infections and illnesses which are thought to occur, or even by the amount of reported illness, it is an important disease in the United States and in most countries of the world. The real human mortality related to this cause is unknown. It is probable that the proportion of infections leading to death is small, and yet the frequency of infections is so large that salmonellosis must have a relative importance which counterbalances its commonly observed minor character.

Although some salmonella illnesses are distinct clinically, many are not. Symptomless infections are common, and the gastrointestinal expression of many infections is in an illness which can only be defined by the isolation of the causative agent. In these illnesses, the human disturbance may be so minor, or so brief, that the victim does not seek medical attention but instead suffers in silence and is thus unknown to the reporting services, where they exist. This must mean that most incidence figures in most areas are incomplete and may more often be an expression of the laboratory and diagnostic services available than of the real human experience. Because of this, unless special studies are carried out, real human incidence is unknown; and geographical comparisons, or changes by time, are difficult to make. However, the reports which do exist are valuable; as well as showing the distribution of salmonella types and being a starting point for field investigations, they suggest that the salmonella picture does vary by local area and that major qualitative and quantitative human changes have been occurring in many areas over the past decade.

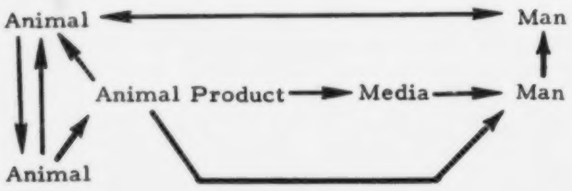
THE EPIDEMIOLOGY OF SALMONELLOSIS

As with most infectious diseases, host and environmental factors play a major part in influencing the level and type of salmonella infections as well as selecting those individuals who will be infected and the severity and nature of their illnesses which may result. These have been well documented elsewhere and will not be described here.

What makes salmonellosis differ from other infections is the dominant role of certain characteristics of the agent in its epidemiology. These are basically simple in nature and are now well understood.

Using the accepted laboratory typing procedures currently in use internationally, the salmonellae can be divided into more than 800 types. These types, although related in many respects, generally differ in their host specificity. This can be demonstrated in the field. For example, a Northern Ireland mixed farm which was exposed to a contaminated feeding stuff containing Salmonella orion, S. infantis, and S. schwarzengrund showed an initial series of infections within its inhabitants. A proportion of the pigs excreted S. orion; the poultry, S. infantis; and the humans, S. schwarzengrund. Later depending upon other factors, mixed infections occurred. But the initial infection resulting from an exposure to small agent doses was largely influenced by a factor in the agent which in practice made it host specific. This specificity is only relative. Most salmonellae can infect most hosts, but the dose of organisms required to cause an infection in the occasional hosts may be very large indeed.

If this observation is placed beside the present salmonella position, a meaningful order can be seen. If the human-adapted salmonellae (S. typhi, S. paratyphi A, B, and C) are excluded, we are left with a large group of food poisoning salmonellae which are adapted to various wild and domestic animals. Their cycle can be shown diagramatically like this:



The basic cycle with each type of salmonella is animal to animal either directly or indirectly. They can pass from animal to man directly, or via an animal product, and they frequently do. However, although this occurs, this rarely results in an infection or an illness in man as the number of

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organisms is usually too small. Similarly, the agents can pass from man to man, but the same factor of dose, limits the frequency of secondary infections.

An intermediate stage is necessary allowing multiplication to take place. This is described as the media stage in the figure. Unfortunately another characteristic of these agents is that many human foods which can be eaten without further cooking are good multiplication media for salmonellae, especially at room temperature.

The complication of this extra step is the probable reason for the relative infrequency of human infections despite the widespread occurrence of these agents. Human infection is a non-essential part of the agents' survival cycle; and, although the path of transfer of the agents from animal cycle to man is clear, man's food preparation habits commonly limit the risks of human infection.

There are exceptions which are at present inadequately explained. Certain salmonellae in some special closed communities apparently can sometimes cause human infections even in small numbers and may possibly be spread from man to man by dust or the airborne route. This may be due to differences in salmonella strains, mutation, selection, or other factors. At present, the recorded outbreaks of this type are rare.

CONCLUSIONS

The usual cycle as described here does give some lead as to possible, and unlikely, methods of control. The prevention of man-to-man transmission by environmental methods or by the control of excreters or carriers is unlikely to result in a large decrease in human salmonellosis.

The education of whole populations about the dangers of certain uncooked foods kept at room temperature is unlikely to be successful in the short term without a major change in food preparation and eating habits, and is impractical.

Certain direct and indirect contacts from animal to man can be made salmonella free as has been demonstrated by the United Kingdom egg legislation of 1963. However, it is improbable that all such contacts can be policed in this way.

Therefore, the most workable and immediate solution must be the selective breaking of the animal cycle starting with the most controllable domestic animals whose specific salmonellae are most important to the human population of a particular geographical area.

I consider that such a selective attack upon the animal cycle is both possible and economical in this country. The host of first choice would probably be domestic poultry.

Identifying Outbreaks

Charles E. McCall, M.D.[1]

Over the past decade and a half, the number of reported isolations of salmonella organisms from human sources has steadily increased. Although this increase in part may be due to better reporting and improvement in laboratory techniques, some of it undoubtedly is due to a rise in the incidence of salmonellosis among the general population. In order for control to be eventuated — be it by health authorities, physicians, hospitals, or industries — the dynamics behind this rise must be better defined by epidemiological investigation. Before epidemiological investigation can progress, the epidemic must be recognized.

THE CHANGING PATTERN OF EPIDEMICS OF SALMONELLOSIS

Concomitant with the rise in the incidence of salmonellosis is an increased difficulty in recognizing an epidemic when it occurs. This difficulty has been precipitated by a change in the pattern of the epidemic in recent years. Frequently, the present day outbreak involves large numbers of people who are exposed to the same vehicle of infection but who do not live in close geographical proximity; and therefore involve more than one physician, more than one local health department, or in some cases more than one State health department. The outbreak often encompasses several towns, several counties, or even several States. This is in contrast to the time when outbreaks were almost always confined to families or communal gatherings. Although the small outbreak is still the most common type, the large epidemic is becoming increasingly important. This large and widely dispersed epidemic is the price we pay for modern conveniences, the mass production and widespread distribution of common food items, and the refinements in transportation.

Several outbreaks may be cited evincing this changing pattern in epidemics of salmonellosis.

In 1962 and again in 1963, there were two large intrastate outbreaks of salmonellosis originating in the State of Washington. Both of these involved over 300 people. In 1957, an outbreak of *Salmonella reading* gastroenteritis involved 317 people in 26 States and extended over a 10-month period. In 1961, an outbreak of *S. hartford* gastroenteritis involved 145 people in 18 States and extended over a 4-month period. In neither of these outbreaks was the vehicle of infection identified. These last two epidemics, in addition to an outbreak of *S. thompson* gastroenteritis in Canada traced to cake mixes, provided the impetus which led to the formation of nationwide salmonella surveillance. One of the objectives of this program was to improve recognition of the outbreak, both on a local and national level.

METHODS OF RECOGNITION

The *sine qua non* of recognizing an epidemic of salmonellosis is prompt and complete reporting by private physicians. Recognition of both the small localized epidemic and the large, widely dispersed epidemic depends upon this factor. The method for recognizing the two varies, however. Epidemics involving a small number of people such as families or groups within a community should be detected by the alert physician, whose duty it is to make a specific diagnosis and report the outbreak to local health officials. Larger outbreaks, however, may involve many physicians and health officials — none having seen or heard of a significant number of cases; and thus the true import of the outbreak may be overlooked.

By definition, an epidemic involves a significant increase in the usual prevalence of a disease over a specified period of time and within a specific geographical area. Recognition of the large outbreak of salmonellosis, therefore, depends upon a base line of specific information about the disease within the area involved, be it a State, region, or nation. This data must be developed by surveillance. The basic information presently in use to establish the norm is age, sex, date of isolation, geographical location, and serotype. Deviations of any one of these parameters from the norm within the area involved serve as indicators of an outbreak. This is best explained by illustrations. Although no large outbreak of salmonellosis has yet been recognized by variations in expected age and sex patterns, one can readily see how variations in these parameters could serve as indicators. In September 1962, an outbreak of S. typhi-murium gastroenteritis in Kansas affected 300 men and women attending a single dinner. The age range was 19 through 64. This resulted in a significant variation from the expected age incidence for Kansas; the modal peak usually occurs in the 1 to 10-year age group.

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Incidence and serotype frequency serve as the most valuable parameters to follow when watching for an outbreak. At the Communicable Disease Center (CDC), these parameters are followed by maintaining a large tally board on a weekly basis. This tally board compares the weekly and monthly isolations of salmonellae from humans reported by all 50 States. A partial reproduction of the board is depicted in Figure I. In addition to being listed separately, States are grouped by geographical sectors, thus facilitating the recognition of a multistate outbreak. Cumulative totals for each serotype and the percentage of total isolations represented by the common serotypes are also calculated each week. If isolates from an epidemic of salmonellosis are reported, the number and geographical location of these isolates, whether the epidemic is intrastate or interstate, will significantly alter prevalences portrayed by the board. Thus, this board supplies a moving picture of the prevalence of salmonellosis throughout the country. It permits recognition of any outbreak of salmonellosis in which the isolations are reported.

As can be seen, an epidemic is more easily and quickly recognized when one or more of the following conditions exist: a rare serotype is identified, a number of isolations of the same serotype are reported from a confined area, or a rapid deviation from the expected is observed. However, any significant deviation, be it among the 10 most prevalent serotypes for the State or country or be it among the rarer serotypes, usually indicates an outbreak, regardless of how small or subtle the variations.

The two interstate outbreaks previously mentioned further illustrate the point. In 1957, the Ohio State Health Department recognized an epidemic of S. reading gastroenteritis within the State by noting the appearance of an unusually rare serotype. Further inquiry revealed that other States were involved, thus uncovering the interstate outbreak.

In 1962, S. hartford suddenly accounted for 2 percent of the total isolations reported to the CDC. Prior to that time, it had been an extremely rare serotype, accounting for only 20 of 28,000 salmonella isolations reported by the Communicable Disease Center between 1947 and 1958. As mentioned, the outbreak eventually involved over 150 people in 18 States. Many more cases undoubtedly occurred.

The most recent large outbreak illustrating the value of the serotype frequency indicator is the outbreak of S. derby gastroenteritis in the northeastern United States. This outbreak will be described in detail in another paper.

It cannot be over-emphasized that recognition of both the local epidemic by physicians and local health officials and the large epidemic by those who compare the reports with past experience, depends primarily upon prompt and complete reporting.

INADEQUACIES IN THE PRESENT METHODS OF RECOGNITION

Inadequacies in the present methods of recognition may be summarized in two words, incomplete and delayed reporting. A recent investigation performed by Louisiana State Health Department and CDC illustrates the point.

During the months of August, September, and October 1963, one parish in Louisiana accounted for between 20 and 30 percent of the total human isolations of salmonella reported to the CDC from the State of Louisiana; whereas, between January and July, this parish represented only 4 to 10 percent of the total reported human isolations. No serotype predominated, but a mixed outbreak was suspected and an investigation followed.

There were four bacteriological laboratories in the parish that handled all stool specimens cultured in that parish and several adjacent parishes. By comparing the names of persons from whom salmonella was isolated and reported to the State Board of Health and to the Communicable Disease Center with those of the four laboratories in the parish, it was found that the State and the Communicable Disease Center received 56 percent of their reported isolations from the States Regional Health Laboratory; 30 percent from a private laboratory; 12 percent from a hospital laboratory; and 2 percent from a small private clinic. When the total number of serotypes isolated per laboratory was compared with the number reported to the State and Communicable Disease Center, the data revealed that only 43 percent of the isolations from one of the laboratories had been reported. The other laboratories reported between 95 and 100 percent of their isolations.

The prevalence of salmonellosis was then determined by combining the data from all four laboratories and listing each isolate by month of culture. These data were compared with the number of isolations reported from the parish and appear in Table 1.

The differences between the reported list and the list comparing all salmonella isolations are evident. Two important points stand out. First, the degree of under-reporting is impressive. Between January and November 1963, only 37 percent of the total isolations from this parish were reported to the State. This varies from a low of 6 percent in July to a high of 89 percent in October. Secondly, a discrepancy between the date of culture and the date of reporting is suggested. In September, more isolations were reported than actually occurred. To document the suggested delay, all individual

Figure 1.–*Salmonella isolations reported to Communicable Disease Center for week ending February 1, 1964*

SEROTYPE	NEW ENGLAND						MIDDLE ATLANTIC					EAST NORTH CENTRAL					JANUARY						DECEMBER					
	Me.	N.H.	Vt.	Mass.	R.I.	Conn.	N.Y.-A	N.Y.-B.I.	N.Y.-C.	N.J.	Penn.	Ohio	Ind.	Ill.	Mich.	Wis.	Total	% of Total	Total	% of Total	Total	% of Total	Total	% of Total	Total	% of Total	Total	% of Total
Typhimurium				17	2	9	23	15	17	1	26	25	3	23	(71)	4	93	299	172	301	253	270	377	290	523	305	416	275
Anatum				1			7	1			1	1	1		1		5		12		13		19		22		21	
Bareilly								1			1						3		3		3		3		5		6	
Blockley	2				1	2	1		1		2	2		2	4	1	4		7		13		20		26		28	
Braenderup				2				1	1														2		4		5	
Bredeney							1	7	2		2						4		8		16		25		32		20	
Cholerae suis																							1		1			
Cholerae-suis var kuns.																	1		3		3		4		4		2	
Derby				㉕	1	⑩	⑭	㉒	⑲	⑨	㊸	⑭	1	⑲	2	3	37	119	66	115	128	137	177	136	230	134	183	12.2
Enteritidis				13		5	9	2	3		5	2		2	7	3	16	51	21	37	40	4.3	57	44	69	40	61	4.0
Heidelberg				8		2			5	1	3	17	1	11	7	11	17	55	39	68	64	48	84	65	119	69	103	68
Infantis	1				1	1	3	6	6		6	7	4	3	6		15	48	27	41	52	55	67	52	82	48	92	61
Javiana																	1		1		2		9		11		12	
Manhattan				1					1		1	1					4		8		15		9		22		28	
Montevideo				3			2	2	1		5	1	1	2	1		7		17		24		31		42		46	
Muenchen											3	1	1				4		7		10		11		16		20	
Newport									3			4	2	5	3		12	3.9	29	51	47	50	59	4.5	70	4.1	88	58
Oranienburg				4	2	1	1	3	3	1	1			3	1		15	48	20	35	39	4.2	48	3.7	56	3.3	37	24
Panama							1				1			4	1		7		10		24		27		27		9	
Paratyphi B				2			1				3				1				4		6		6		10		13	
Paratyphi B var java							2							4		1	3		6		10		13		24		9	
Saint-Paul				1		1	1	1			2	2		7	3		5	16	9	16	15	16	23	18	37	22	39	26
Schwarzengrund											1			1					3		3		3		5		11	
Tennessee				1		1				1	1		1		5	3	9		11		18		26		32		30	
Thompson				4						2	1	4		1		1	7		14		16		20		30		18	
Typhi					1		2	1				2		2	1		7	23	22	38	36	38	41	35	58	34	30	2.0
Typhimurium var. cop.				8						1					1		1		3		3		5		14		16	
Berta												1							1		2		3		4			
Chester									1		2				2		2		4		6		8		8			
Cubana													1		1	1	3		3		3		4		8			
Give								1					1				2		2		3		5		5			
Kentucky											1										1		1		1			
Litchfield								1						1			1		1		2		4		4			
Meleagridis												1							1		1		1		1			
Miami																	1		1		1		1		2			
Mississippi																							2		3			
Poona									1								1		2		3		3		3			
San Diego				2			2	1						1	1		2		5		9		12		13			

Table 1–*Prevalence of salmonella isolations in a Louisiana Parish during 1963 listed by month of culture and by month of reporting to the Communicable Disease Center*

Month (1963)	Total isolations performed	Isolations reported to the CDC
January	16	5
February	15	2
March	21	3
April	29	5
May	20	5
June	30	7
July	18	1
August	36	11
September	24	26
October	28	23
Total	232	88

isolates reported were reviewed; and it was found that delays between stool culture submission date and serotype reporting dates varied between 1 and 7 weeks, as was seen when corrections for reporting and date of culture submission were made.

The large number of different serotypes isolated and the position of their prevalence did not suggest that an uncommon serotype or a single serotype was predominating. The line listings suggested that the incidence of the disease had not really changed. An epidemic was highly unlikely. Immediate and complete knowledge of the isolations would not have given a false picture.

TECHNIQUES FOR IMPROVEMENT

Based on the concept that control of salmonellosis is of major importance to the economic and health welfare of this country, completely defining the salmonella problem on a local and nationwide scale becomes necessary in order that steps may be taken towards realizing this control. Better recognition of the epidemic is necessary so as to hasten complete definition of the problem, and thus hasten control. Although present methods of recognition are valid in theory and have resulted in recognition of many small and several large outbreaks, their efficiency must be improved. Incomplete and delayed reporting is the largest obstacle to overcome. An increased awareness of salmonellosis as a major problem by public health authorities, physicians, veterinarians, laboratory personnel, and industry is needed. These groups must maintain a constant vigil. Public health personnel must properly channel their efforts in investigating salmonellosis. Investigation of every case of salmonellosis on a local level is impractical. Local and State health departments should follow salmonella reports from week to week, comparing them with the established norm for their area. Epidemiological investigations should be directed towards the outbreak rather than towards the individual case. The trees would then not prohibit the forest from being seen. The end result would be control, a condition that cannot exist until the total picture is recognized and adequately defined.

Hospital-Associated Salmonellosis

Edward W. Hook, M.D.[1] and
Donald Kaye, M.D.[2]

During October and November 1959, Salmonella heidelberg was isolated from the stools of 11 patients and S. enteritidis from the stools of 4 patients at The New York Hospital (a 1000-bed hospital). These infections were recognized over a period of about 1 month. All of the patients had major underlying diseases and had been in the hospital for days or weeks prior to the development of intestinal symptoms. It was apparent that these patients had acquired salmonella infections in the hospital.

The outbreak was recognized about 3 weeks after onset. Stool cultures were then obtained from 50 patients on medical and pediatric wards and were found negative for salmonella. Stool cultures were also performed on 99 persons directly concerned with the preparation of food, and salmonella species were isolated from 5. Only one of these isolates was S. heidelberg; the other four represented two additional serotypes (S. newington and S. muenchen) which had not been isolated from patients. A number of environmental and food cultures were negative for salmonella.

The food handlers who were infected were removed from their jobs, and their salaries continued until their stools became negative for salmonella. No further cases of S. heidelberg or S. enteritidis infection were observed in the patient population. The termination of the epidemic cannot necessarily be related to suspension of infected food handlers as it is possible that the outbreak would have subsided spontaneously.

In November 1961, an outbreak of S. taksony infections occurred at The New York Hospital. These infections occurred over a period of at least 3 weeks. The outbreak was first recognized when, within a period of 1 week, six patients on five different wards developed gastroenteritis due to S. taksony. Stool cultures were then obtained from patients on three additional wards. These studies revealed that S. taksony infection was widely disseminated in the hospital, involving 7 to 22 percent of patients on different wards. Stool cultures were also obtained from 105 workers who were directly concerned with food preparation; 19 specimens (18 percent) yielded S. taksony, but no other salmonella were isolated.

Three hundred and fifty samples of food were cultured, and 3 specimens were positive for S. taksony. One isolate was from grilled tomato, and the other two were from separate batches of tube-feeding mixture. The tube-feeding mixtures contained raw eggs, which were assumed to be the most likely source of the outbreak. The two batches of eggs used in the preparation of the tube-feeding mixtures were traced back through the complex chain of supply to 23 farms in Pennsylvania. Cultures were made of pools of 400 eggs from these farms, and no salmonella were isolated. Approximately 50 fecal specimens were also obtained from chickens on each farm and were negative for S. taksony.

The majority of patients and personnel infected with S. taksony were asymptomatic. However, 13 of the 49 persons from whom S. taksony was isolated had gastroenteritis, and one patient with leukemia and granulocytopenia developed bacteremia. The outbreak was also disruptive and expensive for the hospital in many ways. For example, just before Thanksgiving it was necessary to remove from duty about 18 percent of the key personnel in the kitchen. Concomitant with removal of infected personnel, the outbreak subsided.

Since the S. taksony outbreak, no additional clinically-apparent salmonella outbreaks have been recognized at The New York Hospital. However, three additional stool culture surveys have been carried out on the entire staff of the Nutrition Department at about 6-month intervals during 1962 and 1963. In one survey, no salmonella species were isolated from 354 nutrition workers. In another survey, stool specimens from 5 of 316 (1.6 percent) persons were positive for salmonella of 4 different serotypes (S. heidelberg, S. infantis, S. taksony, and S. typhi-murium). In the third survey 23 (7 percent) of the 327 workers gave stools positive for salmonella. Twenty of the 23 isolates were identified as S. tennessee, and the other 3 isolates were

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S. newport, S. derby, and S. oranienburg. At the time of the third survey, stool cultures were also performed on 52 patients on 2 hospital wards; S. tennessee was isolated from 2 asymptomatic patients, apparently indicating spread of infection to the patient population.

The experience cited in the preceding paragraphs has been reviewed because it demonstrates certain problems caused by hospital-associated salmonellosis at one institution over a 4-year period. This experience is not unique; in fact, hospital-associated salmonellosis has been described with considerable frequency. In the last 35 years, there have been more than 65 separate reports of outbreaks of salmonellosis in hospitals (excluding reports of typhoid fever transmitted within hospitals or mental institutions). Many of the reports summarize several outbreaks, and some refer to personal communications which cite additional epidemics, indicating that the number of hospital-associated outbreaks is considerably higher than actually reported. The annual reports on food poisoning in England and Wales for 1958 through 1962 (1-5) show an average of 21 general outbreaks of salmonella food poisoning in hospitals each year. During the period covered by these reports, members of the genus salmonella were the most common cause of outbreaks of food poisoning in hospitals in England and Wales. Furthermore, general outbreaks of salmonella food poisoning were more commonly associated with hospitals than with restaurants, hotels, private parties, or schools.

Sporadic cases of salmonellosis acquired in hospitals have also been described. Black and associates (6) reviewed in detail 26 cases of salmonella infection observed at the Massachusetts General Hospital over a 6-year period and noted that 6 of the 26 patients apparently acquired symptomatic infection in the hospital. Altmann and Gerichter (7) in a 7-year period at a large hospital in Israel noted 86 cases of hospital-acquired salmonellosis. Most of these were sporadic cases; and, in about 50 percent, infection was apparently acquired from another patient with salmonellosis on the same ward.

Additional studies are needed to define the frequency and extent of hospital-associated salmonellosis. It is likely that bacteriologic monitoring of patients, nutrition workers, and other personnel would reveal an impressive incidence of hospital-acquired salmonellosis.

Many aspects of the hospital environment favor the development and detection of salmonellosis. Hospitals are collecting centers for patients with debilitating diseases, many of which alter susceptibility to microbial infection. In a few diseases, there appears to be a "specific" predisposition to infection by salmonella which exceeds any general susceptibility to other bacterial species. The best examples are salmonella gastroenteritis in patients who have had major gastric surgery (8-10), and salmonella bacteremia and osteomyelitis in patients with sickle hemoglobinopathies (9, 11, 12).

The hospital nursery has been involved frequently in outbreaks of salmonellosis. The newborn infant is an unusually susceptible host, and the morbidity and mortality caused by salmonellosis is considerably higher in infants than adults (9,12). Infants also remain convalescent carriers for a longer period of time than adults (9,12), thus providing additional opportunity for dissemination of infection.

Diagnostic and therapeutic procedures such as surgery, antibiotic and steroid therapy, special diets and feedings, and instrumentation of one type or another, must also be considered in the genesis of hospital-associated salmonellosis. Bohnhoff and Miller (13,14) and Meynell (15) demonstrated that alteration of intestinal flora of mice by antibiotics results in an impressive increase in susceptibility to salmonella infection. A similar effect has been suggested in man (6,9,16), but convincing evidence that antibiotics alter susceptibility of man to salmonellosis has not been presented. Special feedings have been responsible for a number of outbreaks of hospital-associated salmonellosis, for example, eggnog made with infected eggs (17) and tube feedings containing contaminated yeast (18). Instruments used in hospitals may also be responsible for dissemination of salmonella. An outbreak of typhoid fever was related to the repeated use of inadequately cleaned enema cans (19), and outbreaks of infection with other salmonella have been related to the use of contaminated resuscitators (20) and a contaminated bottle warmer (21).

Patients who are admitted to hospitals because of salmonella infection, as well as those who develop infection in the hospital, provide another source of contamination of patients, personnel, and the hospital environment. Salmonella may be present in the upper respiratory tracts of infected patients (22, 23), and millions of these organisms may be excreted in each gram of feces (24, 25). Salmonella have also been isolated from the hands of patients (26) and nurses (27). A number of studies have demonstrated contamination of the hospital environment. For example, salmonella have been isolated from bed linens (23, 26), hand towels (28, 29), scrub brushes (30), hand basins (30), a bath thermometer (31), a table (27), scales (27), an infant weighing basket (32), resuscitators (20), a bottle warmer (21), a gavage cart (27), an air sample (27), dust (23, 28, 29, 32), and from hospital mice (28-30), flies (33), and cockroaches (30).

Additional factors which may serve to increase the apparent incidence of salmonellosis in hospitals are the likelihood of noticing minor illnesses in hospitalized patients, the ready availability of facilities for bacteriologic diagnosis, and routine stool surveys of personnel carried out in certain institutions.

The mechanisms of spread of salmonella in hsopitals will not be considered in detail. The majority of recognized outbreaks are explosive in onset, limited in duration, and related to the ingestion of a single item of food contaminated with salmonella.

In these instances, there appears to be relatively little secondary spread of infection from patient to patient. In contrast, salmonella outbreaks in hospitals may follow a different sequence in which infection, once introduced, continues to occur over a period of weeks or months. Spread is apparently not related to a common food source but probably is due to contact involving personnel, patients, or inanimate objects, or is by the aerial route. Outbreaks in which cross-infection appeared to be the mode of transmission of infection have been described predominantly in nurseries or wards for infants (20, 21, 27-32, 34-38). A common chain of events in nursery epidemics is infection of an infant at birth by a mother with salmonellosis and then subsequent secondary spread of infection from infant to infant in the nursery. However, adults are also apparently vulnerable to cross-infection with salmonella as illustrated by the experiences of Datta and Pridie (23) and Close and associates (39).

Summary

Hospital-associated salmonellosis is not an unusual event. Infection is usually food-borne but may be spread by cross-infection involving patients, personnel, or inanimate objects or by the aerial route. Cross-infection has been important as a route of transmission among infants in nursery outbreaks, but may also occur in adults. Additional studies are required to delineate the magnitude of hospital-associated salmonellosis and to document mechanisms of spread.

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State Surveillance of Salmonellosis

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Salmonellosis became notifiable in Washington in 1941. During that year, 84 cases of typhoid fever and 26 cases of laboratory confirmed salmonellosis exclusive of typhoid fever were reported. In 1963, the number of reported salmonellosis cases had risen to 426, and the incidence reached 14.2 cases per 100,000 population. During the 23-year period for which data are available, a period in which the number of recognized serotypes grew prodigiously, an important shift occurred in the distribution of serotypes isolated in Washington. Typhoid and paratyphoid fevers became must less frequent in occurrence, and a large increase in the reported incidence of disease due to serotypes of animal origin occurred (Table 1). The gradual disappearance of typhoid and paratyphoid fevers can be ascribed to continued improvement in sanitation and to the resulting continued diminution of the human carrier reservoir through mortality in the older age groups involved. The growth in incidence of reported salmonellosis other than typhoid and paratyphoid fevers is not as quickly explained, since it may be due, to varying extents, to improved clinical diagnosis, improved laboratory methods, better reporting, expanded investigative activities, or a true increase in incidence. A marked increase occurred in 1963 which is known to be more apparent than real in that a portion of it is composed of cases identified in outbreaks and confirmed through enthusiastic employment of laboratory facilities by investigators. However, there is a significant component of the increase which can be explained only by a true increase in incidence or by an increased level of recognition and reporting.

The surveillance of salmonellosis in Washington involves the regular and systematic collection, collation, and interpretation of reports of laboratory confirmed cases of the disease. The development of a statewide surveillance program was related, of course, to the need for the rapid identification of outbreaks, and it has filled this need frequently. It also permits the surveillant to identify and single out promising areas of investigation through the delineation of seasonal and secular trends in incidence, age, sex, geographic, and serotypic distributions. It may help to identify previously unknown sources of infection and modes of transmission, particularly when a rare serotype is encountered. Under carefully controlled conditions, a surveillance program can also provide a means of evaluating the effectiveness of a salmonellosis control program.

The salmonellosis problem may be likened to an enormous iceberg of which at least 99 percent may be concealed beneath the surface. Salmonellosis is, in the majority of cases, a mild and self-limiting disease. Only a small proportion of cases come to the attention of physicians. Even when consulted, physicians commonly do not obtain specimens for laboratory examination unless the patient is severely ill. Very few patients with salmonellosis are hospitalized, and even hospitalized patients are frequently not given the benefit of laboratory diagnosis. The age distribution of laboratory confirmed cases in Washington during the past seven pericensal years is also evidence of underreporting in that the preschool children, although possibly least likely to contract salmonellosis because of a relatively restricted diet, comprised over one-third of the total (Table 2). This anomaly is undoubtedly related either to a greater severity of illness in the very young or to factors of parental and medical concern which force laboratory diagnosis. It is unlikely that it is related to age selection of exposure to contaminated food substances per se; but it may, of course, be related in part to immunological experience in the younger age groups.

A variety of factors, therefore, tends to obscure and minimize the problem. For example, at a family reunion in Orondo, Washington, in 1961, eight of nine persons who had shared an inadequately baked turkey contaminated with Salmonella typhimurium developed classical salmonellosis. A physician, alarmed at the severity of the illness in the one victim he saw, called to report what he thought might have been typhoid fever. One other family member was seen by a different physician, but no cultures were taken; and, therefore, none of the cases were reported. An investigation uncovered eight cases, all subsequently confirmed in the laboratory.

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Table 1.—*Distribution of prevalent salmonella serotypes isolated from reported human cases in Washington, 1941-63*

Serotype	1941-44		1945-49		1950-54		1955-59		1960-63[1]	
	Number	Percent	Number	Percent	Number	Percent	Number	Percent	Number	Percent
anatum	.....	.....	.....	.....	4	.7	.....	.....	.....	.....
bareilly	.....	.....	.....	.....	13	2.3	.....	.....	.....	.....
cholerae-suis	16	5.7	9	3.4	.....	.....	.....	.....	.....	.....
enteritidis	3	1.1	1	.4	.....	.....	.....	.....	.....	.....
heidelberg	.....	.....	.....	.....	.....	.....	25	3.4	163	17.1
infantis	.....	.....	.....	.....	.....	.....	15	2.0	47	4.9
montevideo	.....	.....	.....	.....	26	4.6	40	5.4	24	2.5
muenchen	.....	.....	.....	.....	.....	.....	42	5.7	.....	.....
newport	.....	.....	5	1.9	11	1.9	53	7.2	21	2.2
oranienburg	.....	.....	.....	.....	20	3.5	14	1.9	.....	.....
paratyphi A	4	1.4	.....	.....	.....	.....	.....	.....	.....	.....
paratyphi B	27	9.6	39	14.9	4	.7	.....	.....	13	1.4
reading	.....	.....	.....	.....	.....	.....	34	4.6	.....	.....
saint-paul	.....	.....	.....	.....	.....	.....	.....	.....	25	2.6
san-diego	.....	.....	.....	.....	.....	.....	.....	.....	17	1.8
tennessee	.....	.....	.....	.....	5	.9	.....	.....	.....	.....
thompson	.....	.....	.....	.....	4	.7	13	1.8	13	1.4
typhi	211	75.4	120	45.8	68	12.1	22	3.0	21	2.2
typhi-murium	9	3.2	8	3.1	98	17.4	238	32.4	408	42.8
Other	.....	.....	.....	.....	27	4.8	108	14.7	108	11.3
Not specified	10	3.6	80	30.5	284	50.4	131	17.8	93	9.8
Total	280	100.0	262	100.0	564	100.0	735	100.0	953	100.0

[1] 4-year period. All others, 5-year periods.

Table 2.—*Age distribution of cases of laboratory confirmed salmonellosis in Washington State, 1941-63*[1]

Age group	Number	Percent
0 - 4	745	37.0
5 - 9	225	11.2
10 - 14	119	5.9
15 - 19	86	4.3
20 - 29	158	7.9
30 - 39	157	7.8
40 - 49	167	8.3
50 - 59	129	6.4
60 and over	225	11.2
Total	2,011	100.0

[1] Excludes 341 cases for which the age was not reported.

Later in 1961, a small child was hospitalized in Ellensburg, Washington, with a diagnosis of severe gastroenteritis ultimately shown to be of S. heidelberg etiology. An investigation showed that 9 of 11 persons present at a party with the index case consumed eggnog and developed nearly identical illnesses. Three had been seen by physicians, but no laboratory specimens had been obtained. As a result of the investigation, eight additional cases were laboratory confirmed.

In 1962, a college student in Spokane, Washington, hospitalized with acute severe gastroenteritis and bacteremia due to S. typhi-murium, led to an investigation which ultimately yielded 238 additional presumptive cases, of which 42 had been treated by the college physician without the benefit of laboratory examination. During the investigation, fecal specimens were obtained from 18 randomly selected students who reported illness; 14 yielded S. typhi-murium. Had the hospitalized patient not been identified through a laboratory specimen a massive institutional outbreak, ultimately shown to have been due to contaminated egg whites used in making meringue pies, might have been overlooked. These are three examples of outbreaks each of which was identified through a single hospitalized case.

Several additional outbreaks have been recognized through investigations which started with only two or three confirmed cases. It is literally true that the epidemiologist, armed only with a handful of reports of recent laboratory isolates of salmonellae and a sagacious mind, can almost invaribly identify a common vehicle outbreak or two without inordinate effort; and it is an unusual investigation, indeed, which fails to identify collateral cases in the process of investigating a "sporadic" case. If the usual practices of food marketing, storage, preparation, and consumption in this country are studied, the reasons for these observations become fairly obvious. Very few food substances are marketed except in lots measured to reach hundreds or thousands of persons. Due to mixing or assemblage, contamination of foods such as eggs, poultry, meat, or bakery products tends to occur in large measure rather than in amounts designed to serve only small numbers of persons. Further, the vast majority of meals are shared, so that usually several persons are more or less simultaneously exposed to the same food or ingredient at a given meal. Even in the so-called "family outbreak" which commonly accounts for 20 to 30 percent of all reported cases, the infection is ordinarily incurred simultaneously through ingestion of a contaminated food substance and person-to-person transmission is much less common in our experience. We are, in fact, impressed with the infrequency of true secondary cases in all but the lowest socioeconomic strata. Clearly, the diagnosis of a case of salmonellosis in a family increases markedly the probability that other cases in the same family will be diagnosed and, therefore, reported. The tragedy is that the food responsible for a "family" outbreak may have caused illness in large numbers of families, even in the same geographic area, and escaped identification due to lack of medical attention, lack of laboratory diagnosis, or lack of investigation.

The recognition of outbreaks as may result from the simultaneous infection of extremely large numbers of persons at a common meal generally presents little difficulty. Even if none of the cases has come to etiologic diagnosis, notification of public health officials is likely to occur, the probability of notification tending to vary directly with the magnitude of the outbreak, of course, and with a variety of other factors. However, the configuration of an outbreak is not uncommonly such that the epidemiologic relationship of the cases is not immediately obvious, and it is in such situations that the importance of continuing surveillance is most readily demonstrated. Such an outbreak occurred in 1962 in Washington, and probably extended into three other States. During the fall of that year, 25 isolations of S. typhi-murium were made from fecal specimens submitted from several hospitals in eastern Washington. The number was clearly in excess of expected levels; but the dates of onset, as indicated on the laboratory request forms, were distributed over a period of almost 4 weeks and indicated, therefore, a source other than a common meal. A disparity in the age distributions of this group of 25 patients and the more usual aggregate of reported cases was obvious and suggested strongly that these cases must have been derived from a different, but definable, subpopulation. A preliminary investigation revealed the following:

1. In only two of the involved households was there more than one case, and in none were there more than two cases.
2. Only two of the cases were in persons under 10 years of age. The majority were in young adults and middle-aged persons.
3. The geographic distribution of cases covered an area of several hundred square miles.
4. A majority of the patients were representative of higher socioeconomic strata.
5. Other than in the two households with two cases each, there was no genetic relationship between the patients.
6. No social relationship existed between the patients, and with the exceptions noted above, they were not at all acquainted with each other.
7. A majority of the patients were socially active individuals who tended to eat away from home frequently.
8. There was no common denominator of occupation or religion.

From these observations it was hypothesized that the vehicle of transmission must have been a restaurant food. The possibility of a single restaurant was quickly dismissed and attention was turned to a food common to many restaurants. Before the first day of the investigation had ended, a definite history of consumption of meringue pie ultimately traced to a single commercial bakery had been obtained from 11 of the patients. This information led to the investigation of a church-sponsored luncheon at which one of the patients ate the pie in question. It was learned that at least 180 persons, residents of many different parts of the State, had attended the luncheon; and although no attendance record was available, it was possible to locate 65 of them by telephone. Thirty provided a history entirely compatible with salmonellosis occurring within 56 hours of the time of eating meringue pie at the common meal. Fourteen had been treated by physicians, but none were reported. None of the victims knew that anyone else was ill.

A list of 136 institutions, restaurants, and distributors in a 4-State area receiving pies during the period corresponding with the temporal distribution of the then known cases was obtained. Ultimately, through investigation of outlets serving definable populations, it was possible to document over 300 probable and confirmed cases of salmonellosis associated with meringue pies from one source. Many times that number probably occurred. Bacteriophage typing of strains of S. typhi-murium obtained from 22 patients established that they were identical, further evidence favoring the common vehicle hypothesis.

Approximately 1 year later, a telephone report of an outbreak of gastroenteritis among students of a private college was received. Fecal specimens from 14 patients in hospitals in seven widely separated communities in Washington arrived in the State laboratory almost simultaneously. Once again these cases and more than 300 additional confirmed and presumptive cases were linked to the consumption of meringue pie from the same bakery. In neither of these outbreaks was it possible to confirm the suspected contamination of meringue used on the pies since none was available for laboratory examination. It was, however, possible to demonstrate that similar cans of frozen egg white from the same producer who supplied the bakery were contaminated with the same serotypes. An embargo was placed on approximately 450,000 lbs. of frozen egg whites, yolks, and whole eggs; and 1,500 core samples from the 30-lb. cans were obtained. Results of a partial examination of these samples is shown in Table 3.

Table 3.–*Results of survey of frozen egg whites, yolks, and whole eggs produced by a single firm in Washington, 1963*

		Number positive	*Percent*
Number of Samples	812	134	16.5
Number of Lots	78	25	32.1

Serotypes isolated

Salmonella infantis
Salmonella schwarzengrund
Salmonella worthington
Salmonella montevideo
Salmonella cerro
Salmonella thompson
Salmonella kentucky
Salmonella heidelberg
Salmonella typhi-murium
Salmonella oranienburg
Shigella sonnei

Because the bakery, the largest of its kind in eastern Washington, has used egg whites from the same supplier for many years, there is an implication that other outbreaks of salmonellosis of identical source may have been overlooked in the past. Investigation of the egg-breaking plant revealed that cracked and dirty eggs made up a substantial proportion of the eggs purchased for breaking. Tracing eggs to production sites in this case was not possible since there are over 20 States in the production area, and records which might identify the contents of a lot had not been kept.

During the investigation of the bakery and egg processing plant, frozen eggs from other sources in Washington and in other States were embargoed and examined. This limited investigation, the results of which are shown in Tables 4 and 5, constitutes the early phases of a statewide surveillance program which ultimately must involve an assessment of the degree to which various food products, animal feeds, and food processing establishments are contaminated with salmonellae.

As implied earlier, it is possible that less than one percent of salmonellosis cases are actually laboratory confirmed and reported. It is also possible that the very low mortality rate from salmonellosis is underestimated. A collation of death certificates of cases of gastroenteritis and laboratory reports indicates that a surprising number of cases have not been cultured. That at least a few of these may be of salmonella etiology is not improbable, and possibly death certificate review should be considered as a surveillance instrument. At present, about one-half of the isolates obtained in our laboratory are associated with voluntary reporting by the attending physician. The remainder are simply self-reported from data obtained from laboratory request forms. What is much more important is the reluctance of physicians to obtain fecal or blood specimens from patients with febrile gastroenteritis or typhoid-like illnesses for bacteriological examination. Hopefully, a gradual improvement in this situation will occur as the enormity and seriousness of the salmonellosis problem becomes better appreciated by the practicing medical profession.

Epidemiologists are loathe to admit that frequently their usual lines of communication are inferior to the news media in identifying outbreaks of salmonellosis, but radio and newspaper reports of the occurrence of "food poisoning" not infrequently turn out to refer to salmonellosis outbreaks. No surveillance program would be complete without assiduous attention being given regularly to every such report. Occasionally, a private citizen may call to report an outbreak which has escaped the attention of local public health authorities and the news media. School absenteeism can serve as a surveillance index, especially if reasons for absenteeism are recorded so that gastroenteritis may be distinguished from respiratory disease and other non-salmonella induced conditions.

Table 4.–*Results of survey of frozen egg whites, yolks, and whole eggs from 7 producers in California, Utah, Illinois, and Nebraska, 1963*

		Number positive	*Percent*
Number of Samples	181	96	53.0
Number of Lots	15	7	46.7

Serotypes isolated

Salmonella infantis
Salmonella schwarzengrund
Salmonella montevideo
Salmonella cerro
Salmonella thompson
Salmonella bredeney
Salmonella kentucky
Salmonella typhi-murium
Salmonella oranienburg
Salmonella tennessee
Salmonella saint-paul
Salmonella heidelberg
Salmonella alachua
Salmonella kaapstad
Salmonella california
Salmonella lexington
Salmonella muenchen

Table 5.–*Results of survey of frozen egg whites, yolks, and whole eggs from 12 producers in Washington, 1963*

		Number positive	*Percent*
Number of samples	106	6	5.7

Serotypes isolated

Salmonella infantis
Salmonella heidelberg
Salmonella typhi-murium
Salmonella montevideo

Surveillance of nonhuman salmonellosis and salmonella contamination is certain to play a large role in the total surveillance program. Numerous surveys of foods and food processing environments have contributed much to an appreciation of the ubiquity of salmonellae and the conditions under which animals are infected and contamination may occur during food processing. However, such studies have not always been closely correlated with the human disease problem; and it is in this area of cooperation between epidemiologists, microbiologists, veterinarians, and food sanitarians at various levels of government that progress may become greatly accelerated.

Large Population at Risk

Fred S. Thatcher, Ph.D.[1]

The potentialities for widespread dissemination of pathogens among a large population when contaminated raw materials are used in the manufacture of non-sterilized foods have been demonstrated recently.

The large population at risk was that of Canada, together with the populace of other countries exposed through importation of the foods in question. The raw materials to be discussed are egg products. The disseminating foods are a variety of processed foods containing egg, or contaminated in the factories by exposure to the dust of powdered eggs, and with special reference to the "convenience foods" known as cake mixes and meringue powder. The former are marketed chiefly in small retail packages, the latter mainly in large containers for use in bakeries, hospitals, and other institutions. Salmonellosis has been a sequel to their use by the public and by institutions.

The records for salmonellosis in Canada as reviewed by Best and Yurack (1) show that salmonellosis increased abruptly during the years 1959 to 1962. During this period, the frequency of isolation of Salmonella thompson from human sources increased from 0.5 percent of the total isolations to 25 percent. S. heidelberg also demonstrated a dramatic increase in frequency of isolation. Within this period, S. thompson was reported from human cases of salmonellosis across the 4,000 mile breadth of the country. For the most part, cases occurred sporadically. This pattern of occurrence suggested some widely disseminated source.

As soon as this trend was noticed, the laboratories of the Food and Drug Directorate began a systematic examination of foods with special emphasis on foods of recent popularity. Packaged "cut-up" chicken meat and frozen broiler carcasses were examined in 1959. Fifteen percent were shown to be contaminated with salmonellae, but S. thompson was not found at that time (2). Frozen and powdered egg products were then examined. Some 20 percent were shown to contain salmonellae, a proportion that was later increased by application of an improved method of isolation (3). S. thompson was among the 8 serotypes recovered from 117 specimens.

Canadian retail stores market scores of foods containing egg products. Analysis has been carried out from a representative sampling of all of them. Among these, two types were found to be very frequently contaminated, viz., cake mixes and meringue powder. During the first half of 1961, 61 of 119 cake mixes were shown to contain salmonellae — a frequency of 51.2 percent. Salmonellae were recovered from 60 percent of the cakes containing egg white, and from 40 percent of those containing other egg preparations. Several mixes contained more than one serotype; but, among all mixes in which salmonella was found, 70 percent contained S. thompson (4).

Table 1 lists the many foods containing egg, from which salmonellae have been isolated by this Directorate. The various "convenience" mixes are most commonly involved.

Table 1.—*Foods containing egg and contaminated with salmonellae*

- Cake mixes (25 kinds)
- Cake-frosting mix
- Pancake and waffle mixes
- Meringue powders
- Whip-powder mix
- Batter mix
- Pie crust mix
- Tea biscuit mix
- Cookie mixes:
  - Chocolate chip
  - Butterscotch
  - Brownies
- Biscuit mix
- Gingerbread mix
- Doughnut mix
- Sponge pudding mix
- Coconut dessert mix
- Baby-food mix: Vanilla custard
- "Fondant" bakery ingredient
- Ready-to-bake dough:
  - "Refrigerator rolls"
  - "Icebox cookies"
- Egg barley
- Frozen cake (3 varieties)
- Frozen dessert pies
- Childrens' toy-set cake mixes

EPIDEMIOLOGICAL INVOLVEMENT OF EGG PRODUCTS

Egg products had been known for years to be a frequent source of salmonellae (5). Because cake mixes were not known to have been authentically involved in human disease, and because all salmonellae present in selected mixes were shown to be destroyed when the product was prepared and cooked in accord with the manufacturers' recommendations (4), the importance of our finding in relation to salmonellosis remained uncertain. Accordingly, all provincial health departments were asked to aid in securing epidemiological evidence in cases of salmonellosis to determine whether they could be related to the use of cake mixes. In the meantime, discussions were held with representatives of the industries concerned, with a view to regulatory control.

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The first epidemiological response was received from our easternmost province, Newfoundland. Details have since been published (6). Eleven cases of confirmed S. thompson occurred in a small community, and eight unrelated cases were noted sporadically during 1961. During the three previous years, S. thompson had been isolated in Newfoundland only once. Local eggs, hen droppings, and frozen egg melange used at bakeries were all negative for S. thompson. Cake mixes of the brands used in the homes concerned were positive.

Three of 10 sporadic cases involving young children were investigated in 1962. In each of the homes involved, cakes had been prepared, within a few days before the infections were recognized, from packaged cake mixes bearing the same lot numbers as those later shown to contain S. thompson. One case was fatal (7).

In February 1962, S. thompson was isolated from the stools of a seriously ill child. Stools from all other members of the family and of recent visitors were negative. An unopened box of chocolate cake mix, bought at the same time as one baked a few days before the child was hospitalized, contained S. thompson. Others from the local store contained S. thompson (8).

A few months later, another child, from the community first referred to, developed diarrhea, vomiting, and later, convulsions. Meningitis was diagnosed. S. heidelberg was isolated from the spinal fluid. Locally-sold cake mixes contained S. thompson and S. heidelberg. In another Newfoundland incident, a hospitalized child was reported by its mother to have licked the bowl in which a cake mix had been prepared, a "day-or-two" before the child became acutely ill. S. thompson was isolated from mixes of the same lot number. Other members of the family revealed no pertinent findings (9).

These reports did not constitute proof of involvement of cake mixes, but the circumstantial evidence was considered sufficient to warrant recall from the market of all lots of cake mixes known to contain salmonella, and to recommend promulgation of a new regulation of the Food and Drugs Act, "No person shall sell any egg product for use as food unless it is free from the genus Salmonella, as determined by the official method." The regulation became effective on June 1, 1962, after prior discussion with representatives of the egg products industry and with the cake mix manufacturers involved. Both groups strongly endorsed the proposed action. Mandatory recall has been required for every lot of processed food in which salmonella was positively identified. In the meantime, outbreaks involving S. thompson were reported to have occurred within at least four hospitals. Cake mix and meringue powder were involved. In two carefully studied outbreaks in the Pacific coast Province of British Columbia, S. thompson was found in "bulk" commercial cake mixes from which patients' food had been prepared, and also in specimens from several sites within the hospital kitchen and food storage areas, where the "mix" had been handled (10). S. thompson was recovered from several patients suffering from salmonellosis.

A small outbreak involving several workers at a highway camp in Saskatchewan is documented by Skoll and Dillenberg (11). This was linked to packaged cake mix of the "angel-food" type. S. thompson was recovered from the patients; from a cooked, partly eaten cake; from an additional package of cake mix at the site; from other cake mixes of the same lot number; and from the same store, from whence came the packages found at the camp. The investigators found no evidence of a carrier at the camp. The milk, water, refrigeration, and kitchen sanitation were above reproach.

A similar episode involving meringue-topped pie at an oil-refining site in Saskatchewan is described by Casey (12). S. thompson and S. typhi-murium were isolated from meringue; S. typhi-murium was also isolated from patients.

EGG-CONTAMINATED FOODS IN HOSPITALS AND INSTITUTIONS

Because of involvement of hospitals, special efforts were made by the Directorate, through the Dominion Council of Health and the epidemiologists of the Department of National Health and Welfare, to alert all hospitals to the need to destroy all stocks of egg powder or egg-containing dessert mixes unless they were shown, by laboratory examination, to be free from salmonella. Some 3 months after promulgation of the egg products regulation referred to above and after much attendant publicity, the Food and Drug Directorate examined the kitchens of 50 hospitals. Thirty of them were using commercial egg-containing mixes of ages ranging up to 10 years. Samples from 19 of the 30 yielded S. thompson, often associated with S. heidelberg.

An error by a member of a hospital kitchen staff, who did not know that meringue powder contained powdered egg white, and, consequently, had not disposed of old stocks of meringue powder, led to an explosive outbreak among staff and patients of a hospital in Alberta (13). Of 116 cases reported, salmonellae were isolated from stool specimens of 74: 59, S. thompson; 10, S. heidelberg; and 5, both serotypes. Both serotypes were recovered from the mix, and at a combined density of 7 million organisms per gram. The meringue had been served as a topping for custard pie.

More recently, officers of the Department of Health of the Province of Ontario have kindly provided details of serious outbreaks in their province during 1963 (14, 15, 16). This courtesy is gratefully acknowledged. The use of commercial meringue powder as a topping for pies in a large institution gave rise to more than 700 cases, including those shown to develop as a result of patient-to-patient contact. The attack rate for the entire community was 48.4 percent; among staff members, 7.2 percent; and among food-handlers, 73.3 percent. The serotype recovered from the patients was S. thompson and, in a few instances, S. heidelberg. Both serotypes were present in the incriminated mix. The hospital storage areas and kitchens were shown to be severely contaminated with salmonellae.

SPORADIC CASES FROM EGG-CONTAINING FOODS

Widely dispersed small outbreaks have also been traced to a common source, egg meringue prepared by two manufacturers but containing powdered egg white from a single factory. Hundreds of cases were believed to have resulted from distribution of this "bulk" product to bakeries, where it was used as topping for pies, cakes, and other desserts. The meringue contained S. thompson and S. typhi-murium. By application of phage typing techniques to the latter, the common involvement of many dispersed outbreaks was indicated. Additional cases were also reported and shown to have arisen from packaged products to which no powdered egg had been added. These had become contaminated within one of the factories using powdered egg white of the same source involved in the other Ontario outbreaks described above. The serotypes recovered from patients were also found in the food which they had eaten, and in the dust from the processing area in the factory. White dust abounds wherever powdered egg white is in use.

These serious outbreaks in which the cause has been positively identified are probably not the only instances of disease attributable to contaminated egg products. In 1961, a large hospital for children noted a substantial increase in the number of admissions for salmonellosis. This was associated with a marked increase in the proportion of cases attributable to S. thompson, a condition which definitely subsided during 1963 (15). Other hospitals have records of infants who were carriers of S. thompson and of S. heidelberg.

The seriousness of the problem is clear. But so far as can be determined, all known Canadian outbreaks involving several or many people, and attributable either to cake mixes or to meringue powder, whether in hospitals or among the public at large, contained powdered egg white from a single factory. For the most part, and possibly in all instances

Table 2.—*Salmonella in egg products and in foods containing egg products – 1961–63*

Egg products and foods containing egg products	Salmonella recovery in specified inspection periods														
	Before 1-1-61[1]			6-1-61 to 6-1-62[2]			6-1-62 to 12-31-62[3]			1-1-63 to 6-1-63			6-1-63 to 12-31-63[4]		
	Number tested	Number positive	Percent	Number tested	Number positive	Percent	Number tested	Number positive	Percent	Number tested	Number positive	Percent	Number tested	Number positive	Percent
Frozen whole egg	114	27	23.7	6	0	0	1,398	68	4.8	1,289	69	5.3	824	19	2.3
Frozen egg yolk	13	0	0	10	4	40.0	261	4	1.5	199	5	2.5	131	5	3.7
Frozen egg white	9	0	0	3	0	0	231	10	4.3	298	3	1.0	50	10	20.0
Powdered whole egg	...	..	....	56	10	18.0	55	0	0	54	15	28.0	52	6	11.5
Powdered egg yolk	...	..	....	16	2	12.5	110	16	14.5	151	5	3.3	159	0	0
Powdered egg white (plus albumen).	3	3	100.0	89	27	12.5	135	17	12.6	127	13	10.2	107	5	4.7
Total egg products	139	30	21.6	180	43	23.9	2,210	115	5.2	2,118	110	5.2	1,323	45	3.4
Meringue powder (bulk)	...	..	....	3	1	33.0	38	16	42.1	34	13	38.1	20	7	35.0
Cake mixes containing egg.	119	61	51.2	327	100	30.0	371	105	28.3	433	14	3.4	362	[5]10	2.7
Other bakery products containing egg	16	0	0	55	5	9.1	64	4	6.3	19	1	5.5	60	0	0
Dessert products	20	4	20.0	33	1	3.0	66	0	0	38	7	18.4	32	0	0
Other foods containing egg	60	0	0	80	0	0	204	0	0	179	0	0	....	...	...

[1] Before formal contact with industry.
[2] Discussion and educational action pending epidemiology.
[3] Regulation prohibiting sale of egg products if contaminated with salmonella promulgated June 1, 1962.
[4] Period of biased sampling (i.e. against suspect manufacturers).
[5] Includes 5 childrens' toy cake-mix sets.

Table 3.–*Salmonella serotypes isolated from cake mixes and from egg products*

Serotype	Cake mixes and meringue				Egg products			
	June 1961-62	June 1962 to December 1962	1963	Total	June 1961-62	June 1962 to December 1962	1963	Total
thompson	108	113	37	258	16	40	32	88
heidleberg	22	24	3	49	......	7	25	32
typhi-murium	1	1	10	12	......	4	6	10
tennessee	18	18	3	39	28	6	15	49
oranienberg	11	2	....	13	7	3	23	33
bareilly	3	5	....	8	2	2	7	11
saint-paul	......	....	....	....	......	....	....	....
braderup	......	....	....	....	......	1	2	3
senftenberg	......	....	....	....	......	....	5	5
montevideo	12	....	....	12	3	12	3	18
infantis	8	....	....	8	......	10	1	11
pullorum	......	....	....	....	10	1	7	18
worthington	2	....	....	2	1	11	15	27
kentucky	1	2	....	3	1	....	2	3
muenchen	3	3	....	6	......	....	2	2
manhattan	6	2	....	8	3	1	12	16
newington	......	4	....	4	......	2	....	2
give	2	....	....	2	1	....	....	1
enteritidis	......	....	....	....	......	....	....	....
minneapolis	......	2	....	2	......	1	....	1
halmstad	......	....	....	....	......	....	2	2
bredeney	......	....	....	....	......	....	1	1
derby	......	....	....	....	......	7	....	7
arizona	......	2	....	2	......	1	....	1
Frequency of:								
thompson	55%	63%	70%	....	22%	36%	20%	....
heidleberg	11%	13%	57%	....	......	6.3%	16%	....
typhi-murium	0.5%	0.6%	19%	....	......	3.6%	4%	....

except that described below under "tolerances," the incriminated egg products were manufactured prior to the promulgation of the pertinent egg regulation. This factory uses the pan-dry method of drying the liquid egg white.

The effect of exportation of products from this contaminated source to other countries is unknown. It is known that certain shipments of egg white, carrying the same lot numbers as those supposedly under detention in Canada, found their way to England. Fortunately, some of these were rejected by the British health authorities. At least one consignment of angel food cake mix originating in the United States was also rejected by the United Kingdom. Comparably, Canada has rejected, because of salmonella contamination, a number of shipments of processed foods containing egg and egg products manufactured in the United States.

THE IMPACT OF A CONTROL REGULATION

The developments described have occurred in spite of withdrawal from the market of cake mixes valued at hundreds of thousands of dollars and the impounding of tons of egg products. However, since promulgation of the egg regulation, there has been a steady lessening in the degree of salmonella contamination of egg products and of processed foods containing egg (Table 2).

During the year immediately preceding promulgation (a period of extensive "educational effort"), tests of 180 specimens of all types of egg products revealed a mean contamination rate of 23.9 percent. Within 6 months after promulgation (June 1, 1962), an abrupt decline to a contamination rate of 5.2 percent was noted. This remained unchanged in the following 6 months; but, during the second half of 1963, the rate was reduced to 3.4 percent, in spite of bias due to intensified sampling of brands known to be more prone to contamination.

The data in Table 3 shows that S. thompson was the most commonly isolated serotype during all periods and from both egg products and cake mixes. Among mixes, its frequency ranged from 55 percent to 70 percent of all serotypes recovered from such foods; among egg products the frequency ranged from

20 percent to 36 percent. The powdered egg products were the most frequently contaminated.

During 1963, products from the incriminated factory were found to contain the serotypes S. thompson, S. heidelberg, S. tennessee, S. muenchen, and S. typhi-murium in 16 of 29 production lots. All but one of these contaminated lots was manufactured during the first 3 months of the year.

TOLERANCES FOR SALMONELLAE IN FOODS?

A specific experience supports the contention that no tolerance of salmonellae in foods is warranted. When examining egg products at a factory, this Directorate routinely obtains samples from a number of containers (drums, pails, cans, etc.) equivalent to the square-root of the total number of containers in each "lot." Where only one container is found positive for salmonellae, in order to give the owner the "benefit of the doubt" lest the highly improbable event of laboratory contamination could have occurred, it has been the practice to retest all specimens before requiring continued detention of the product and to verify the presence of salmonellae in at least one container by the second examination. In one instance, a second examination failed to show salmonella. A resampling was carried out, found again to be negative, and the shipment was released for sale. It was assumed that this preparation must have contained a very low density of salmonellae, if any. Nevertheless, a "lot" of cake mixes, clearly implicated in disease, was said to have been manufactured from the specific released shipment of egg white.

SPECULATIONS ON THE ORIGIN OF SALMONELLAE IN EGG PRODUCTS

The cause of the contamination of Canadian egg is not completely known. It is most severe in the central prairie provinces. Bigland (17) indicates the probability that poultry chicks, originating from specific hatcheries, represent a major channel for distributing salmonella carriers to poultry farms. Eggs from infected flocks are the source for the hatcheries.

The infected flock which gives rise to internally contaminated eggs may be the chief cause of contamination in egg products. This laboratory has isolated S. thompson from the contents of shelled eggs while failing to find salmonellae on the surfaces of the same eggs. Further, a statistical study of the relationship between fecal organisms, standard plate count, and the presence of salmonellae in egg products and foods containing them, established no statistical correlation between the presence of salmonella and either of the other parameters (4). This suggests that fecal contamination of shells is not the most important source of salmonellae in egg products. With regard to S. thompson, the writer is informed by research veterinarians of the Canada Department of Agriculture, that it is their opinion that chicks which survive an exposure to S. thompson tend to become carriers with localization of the pathogen in the ovaries. Therefore, eggs become contaminated before deposition of the shell, and fecal droppings may not contain the organism. This may have a bearing on the statistical relationship mentioned above.

Another important factor in contamination of foods and the infective sequellae, is the well-established occurrence of cross-contamination within a factory so that products containing no intentionally added egg may, nevertheless, be positive for salmonellae, and may cause disease. The Ontario study reported above is illustrative. In addition, this Directorate, more than a year before the Ontario outbreak, had identified salmonellae in specimens taken at the same factory as involved in that outbreak, in dust from several sites near the mixing machinery, and 10 days after use of powdered egg. Structural changes and thorough cleaning were carried out in consequence. Subsequent tests were negative. New preparations of egg white were later brought to the plant. This led to extensive environmental contamination, causing salmonellosis from foods in which egg was not an ingredient. Comparable cross-contamination has been demonstrated by this laboratory on two other occasions. In each instance, the serotypes found in the nonegg foods were identical with those of the environmental contaminants and the original powdered egg white source. Professional staff of some of the larger milling establishments where cake mixes are made, have described the severe difficulties encountered in maintaining the plant area and machinery sufficiently free from salmonellae to permit preparation of nonegg mixes free from salmonellae.

The demonstrated ease of cross-contamination within factories probably has a counterpart within kitchens, and may well explain the presence of salmonellae on cakes which appeared normally cooked (and hence presumably freed, while in the oven, from contaminating salmonellae). The likelihood of secondary contamination of other foods within a kitchen is obvious.

ADDITIONAL EPIDEMIOLOGICAL NEEDS

Our recent experience points to additional needs for epidemiological investigations. The factory that had disseminated so much powdered egg white contaminated with S. thompson and S. heidelberg, has, during the past year produced egg white contaminated with S. typhi-murium. Its precise source has not been determined. The upward trend for this serotype in human disease is pointed out by Best and Yurack (1). Salmonella worthington was isolated by our laboratories 15 times in 1963. Initially it was found only in frozen whole egg from a plant in a city in the Province of Manitoba, but it was recently found in a second plant. Some preparations of frozen egg have remained positive for S. worthington after being reprocessed three times. None of the known contaminated specimens had reached the market. It is obviously desirable to find and to confine this source. Salmonella worthington was identified in April 1962, from a cake mix purchased in Vancouver, and in the same year, from a frozen cake imported from the United States.

SECONDARY FACTORS INFLUENCING CONTROL

In spite of recent setbacks in control, now that it is recognized that much of the recent Canadian salmonellosis has been caused by egg white from a single factory (unfortunately, a major producer), the Directorate can better focus its attentions on this particular source. A major handicap to control has been the continued use, even by hospitals, of products sold before promulgation of the regulation. Only a few pounds of egg white have been involved in the major outbreaks. It is fortunate that many thousands of pounds of contaminated egg products were prevented from reaching the public.

A further important impediment was the lack of testing facilities competent for analysis of salmonellae. This was acutely true among the egg products industry, but was also unsatisfactory on the part of many manufacturers of processed foods. Competent independent analysts were too few. The Directorate's control program has greatly stimulated the necessary development in this area.

Policy does not permit the Directorate to serve as a control laboratory for all food factories. Many production lots were sampled by the Directorate. These were withheld from sale until a negative salmonella finding was reported, but other preparations reached the secondary processor without adequate sampling and testing, either by the initial manufacturer or by the secondary processor.

CONCLUSIONS

It is the writer's opinion that the disease reported in this paper clearly illustrates the need for more extensive laboratory testing by the food industry in Canada. If small factories cannot meet the cost of testing, then private testing facilities should be encouraged by organized patronage from the manufacturers, possibly through their trade associations.

Canadian experience has shown that widespread salmonellosis can result, throughout the breadth of a continent, if a single source of contaminated raw material finds its way into the giant maw of the modern mass-producing "convenience food" industry. Exportation of such products extends the population-at-risk to a very large one indeed. It illustrates one of the most taxing problems in public health food control: problems which are almost certain to increase until more effective bacteriological control testing becomes an integral part of food processing procedures. The writer would like to give emphasis to a recommendation to this effect made by the International Committee on Microbiological Specifications for Foods (5).

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Panel Discussion

Dr. Kenneth W. Newell, Chairman,
Dr. Ernest A. Ager, Dr. E. W. R. Best,
Dr. Philip S. Brachman, Dr. W. Charles Cockburn,
and Dr. Joan Taylor

DR. NEWELL: The questions to be discussed by the panel have been submitted by the conference participants; and, just as we appreciate the questions from the floor, we would also appreciate comments or answers from anyone who has something to add to the discussions.

> QUESTION: Please comment on new regulations proposed for controlling salmonella contamination of eggs produced in or imported into the State of Washington. Also, how will these regulations be enforced?

DR. AGER: The new regulations in Washington set standards for the production of eggs and egg products. Our old regulations defined salmonella contamination as a form of adulteration and so there is nothing new in this.

It is our feeling, however, that a good deal needs to be done at the production sites, that is in the egg-breaking establishments; and the Washington State Department of Agriculture has adopted a new set of regulations in order to set these standards. We hope there will be a vigorous enforcement of our food adulteration regulations so that we can look for a significant lowering in the degree to which egg products are contaminated.

When these regulations are put into force, frozen eggs can be imported into Washington only if the eggs were produced under conditions quite comparable to those being demanded of our Washington producers. Mr. Cameron Adams of the Washington State Department of Agriculture is in the audience and perhaps he could comment further on this.

MR. ADAMS: Concerning the importation of eggs into Washington, the regulation which has been signed will become effective the first of April 1964 with an additional 60-day period for clearing stocks on hand. The order is based on the same general requirements that the United States Department of Agriculture (USDA) applies to inspected and shielded eggs in those plants under continuous inspection, with some modification of the quality standards for eggs purchased for breaking.

Eggs will be allowed to come into the State without question if they are produced under USDA continuous inspection. This is the only mechanism that we know of by which we can, at the present time, administratively control the importation of eggs from other States. If upon examination, these eggs are found to be contaminated, they will be handled in the same manner as eggs produced within the State.

> QUESTION: Please discuss environmental sepsis due to salmonella in hospitals, and cite evidence for survival of organisms in dust, on fomites, etc.

DR. TAYLOR: Some work has been done by Dr. M. T. Parker (formerly Director, Public Health Laboratory, Manchester) on the question of environmental contamination as related to the age of the patients involved.

It was found that the surroundings of a child up to the age of two tended to be the most highly contaminated, and the degree of contamination gradually decreased as the age of the individuals studied increased. It seems to me that the situation with young children, expecially those less than 1 year of age, is that there is little or no control of the feces and the diaper. This is comparable in some respects to a situation of staphylococcal contamination of wound dressings in hospitals in which there develop uncontrollable environmental contamination and cross infection with staphylococci. Concerning the survival of organisms in dust, all I can quote is the prolonged hospital outbreak which was referred to by Dr. Edward Hook (page 47) in which they checked the dust in a vacuum cleaner and recovered salmonella. They kept the dust in the original bag, and 18 months after the original isolation, the dust was still positive.

QUESTION: In hospital-associated salmonellosis, does the present evidence support infected food or sick patients as the most important source of infection?

DR. HOOK: As to the relative importance of infected food and sick patients, I think, in terms of the total number of outbreaks, that the bulk of the evidence would favor food as being the most common source of hospital infections. But concerning the nursery outbreaks, I would say dissemination by other means is more significant.

DR. TAYLOR: I have carefully reviewed the records of the reported hospital infections in the United Kingdom and have come to the conclusion that, most frequently, a single patient introduces the infection. What happens is that a patient is admitted with loose stools which do not cause any concern, and then three or four more patients become infected. The cases build up, but the whole thing is not spotted for 4 to 5 weeks. It is only by going back over the records most carefully that one gets the impression that it is the admission of a so-called sporadic case, usually a child, that initiates the hospital epidemic.

DR. HOOK: I certainly think there would be no disagreement if you are speaking of outbreaks among infants and young children; but, if you look for that type of secondary spread in outbreaks involving adult wards of general hospitals, they are rare. At least, this seems to be the case with reported outbreaks that are described as single entities.

QUESTION: Dr. Hook, in your opinion, concerning the causes of Salmonella tennessee infection in your hospital, was the vehicle of infection eggs, or some other source, such as the nutritional workers?

DR. HOOK: In all of the instances in our hospital, we were never able to show the precise source of the outbreak.

DR. NEWELL: There are quite a number of questions about what you should do in a hospital if there should be an egg-associated outbreak or what should be done to prevent egg outbreaks. This is the phrasing of one:

QUESTION: What can be done to reduce the potential hazard of eggs introducing salmonella organisms into hospitals?

DR. TAYLOR: I think that this may be accomplished by limiting the use of eggs to the shell egg, and additionally, to shell eggs used on the day they are broken. In this way, one might cut down on the problem of contamination very markedly.

DR. STEELE: May I add a comment to that. Some of the American bakeries that are producing high quality products have found they can buy eggs that are salmonella free. Miss Margaret Lally of the Institute of American Poultry Industries, has shown me her data derived from checking eggs daily; to date, she has not recovered salmonella from grade A shell eggs.

It seems to me that this regimen of buying only quality products would be a very logical procedure to follow for any hospital. I think the same thing would also apply to all other food products of animal origin that hospitals are buying.

QUESTION: How should a salmonella surveillance program be set up in a large city with population greater than 750,000 people?

DR. AGER: I would think a community of that size should have a salmonella surveillance program consisting of essentially the same elements as a State surveillance program. There are a number of considerations for this type of surveillance program. First, I think it is important to encourage the practicing medical profession to make liberal use of the laboratory for the identification of salmonella in gastrointestinal and typhoid-like illnesses.

Every confirmed case should be used to locate collateral cases. I do not think there is any such thing as sporadic salmonellosis. If good surveillance and investigative programs are to be maintained, every case should lead the investigators to other cases.

One method which has helped us at times is the surveillance of school absenteeism, when it is reported by types of illness, such as respiratory and gastrointestinal illness. Such reports often reach local health authorities promptly. School lunch programs more than once have been responsible for significant outbreaks of salmonellosis.

I think it is important to provide practicing physicians with reports of all investigations in which they might have even casual interest. When we ask a physician, "May we interview your patient?" he is entitled to a return report. It is true that not all our reports are read, but many of them are read with great interest. The whole surveillance program receives a "transfusion" every time a group of practicing physicians becomes involved in one of these investigations.

Close surveillance should be maintained of trends in age and sex distributions, geographic distribution, temporal distribution, and any other kind of distribution which might ultimately lead to the recognition of a common denominator. I also think there ought to be close communication with the State surveillance program and, therefore, with the Salmonella Surveillance Unit of the Communicable Disease Center, as a matter of keeping abreast of developments and being able to contribute to those developments.

DR. NEWELL: I have a question which I would like to direct to Dr. Charles Cockburn because he spent nearly 3 years trying to teach me this particular thing.

> **QUESTION:** Should a health agency wish to start reporting salmonella incidents, what terms would you recommend they use? For example, "incidents," "episodes," "family episodes"? Have there been any difficulties in this country or elsewhere in reporting salmonella "incidence" or "incidents"?

DR. COCKBURN: As for the question, "Has there been any difficulty in reporting salmonella incidence?" Of course there is difficulty. This has been made quite clear today. Nobody, in fact, knows what the incidence or the prevalence of salmonella infection is. I think it is quite impossible to consider commencing any surveillance scheme without a good laboratory service, because you must be able to get your specimens examined freely, without any hindrance such as payment for examinations or great distances between health department and laboratory, and so on.

The second requirement is, if surveillance is on a national scale, you should have agreed on terms to use before you begin. What we must do now is to get down not only to national terms but to international terms and to agree upon them because it is apparent today that Canada, the United States, and the United Kingdom are using the same terms in different ways, and this can lead to much confusion. So, you need laboratories, and you need to define terms. It does not matter how you define terms as long as you define them.

Should one use the term "incidents or episodes"? It does not matter whether you call it an episode or an incident so long as you have decided what you mean by these terms and so long as your health officer is able to go out and make an investigation and make a reasonable report on the subject.

DR. NEWELL: I would like to begin with another series of questions. The first one is directed at me.

> **QUESTION:** If control of the disease in man is our aim, what information concerning salmonella infection (or disease) in wild and domestic animals and birds might contribute most meaningfully to such control?

DR. NEWELL: Now this is very difficult to answer specifically and clearly. I think that first a number of judgments have to be made concerning which are the important salmonellae to man. I think we can only judge this by knowing what serotypes have caused illnesses within a particular area or region. From this we have one judgment, that is, which are the important serotypes as far as man is concerned.

We have another series of judgments which we must also make concerning which animals and birds and which cycles are accessible to control. There are some animals in which it would be very difficult to control salmonellosis, but these in general are only related to a very small proportion of the serotypes which are important in human infection. We can probably narrow our considerations down to those animals or birds who, in fact, participate in a salmonellae cycle important to man.

Related to the last question is another one not directed specifically to anyone on this panel but related to a veterinarian, someone on the floor who might be prepared to answer this.

> **QUESTION:** Could a veterinarian at the conference discuss briefly the economic aspects of salmonellosis as related to the farmer, rancher, and poultry producer?

DR. DARBY: In response to this question, I think we must admit that specific information is presently not available concerning the over-all economic losses due directly or indirectly to salmonellosis. At the present time, surveys are being conducted by the USDA to answer this question.

DR. WALKER: In the United States Department of Agriculture, we have wondered what salmonella infection in poultry costs the livestock, poultry, and other industries. Statisticians have helped try to work out a dollar value of the loss; however, our knowledge on which to establish a figure is limited. We know, for example, that the present pullorum/typhoid control program costs participants in the National Poultry Improvement Plans $10,000,000 a year. Other than this known cost, there is not enough information in other areas for us to come to any conclusions. Statisticians suggest we learn more about the incidence of salmonellosis. So the best thing I could suggest is that we gather more factual information to get a truer picture of the situation.

DR. NEWELL: The next series of questions have more to do with illness or are specifically related to the agent or groups of agents.

> **QUESTION:** Can differences in virulence of different species of salmonella be accounted for? For example, S. cholerae-suis infections of humans are more likely to be fatal than

> most other salmonella infections. Secondly, is host-specificity of some species of salmonella an expression of differences in virulence? And thirdly, are there any non-pathogenic (i.e. non-virulent) species of salmonella?

DR. TAYLOR: I think this is an extremely broad question, and I am going to answer it from an odd angle, because when it comes down to a question of host-specificity, I think we are going into what I call high powered scientific bacteriology. One thing we have learned quite recently is the reason why Brucella abortus has a predilection for the placenta. It is because of a metabolic requirement which the bacterium finds in that placental area. I cannot help feeling that a similar circumstance may account for the truly host-specific types of salmonella. In other words, the typhoid bacillus likes something in man, and S. cholerae-suis likes something in the pig. Other salmonella serotypes are not so specific in their metabolic requirements, and thus do not have narrow limits of host-specificity.

Now the question of S. cholerae-suis in man; whenever human infections with S. cholerae-suis occur in the United Kingdom and the United States, it is commonly a fatal illness, and usually there is only a single case. I have always wondered whether the host — that is the particular patient — may not have something to do with the fatal outcome. This is a wild guess, but it is based on the fact that if one looks through the literature for human outbreaks of S. cholerae-suis, they are extremely difficult to find. The only two which I could find were in the Polish literature and are quite well written up. These two were typical food poisoning outbreaks, and the patients did not die.
Concerning the question as to whether there are any non-pathogenic salmonella, I do not know. In this respect, I always refer to the outbreak of S. tennessee infection which occurred in the premature nursery of a general hospital in the southern part of the United States and which was reported by Dr. Watt (1). Many infants were infected, and the environment was contaminated. The nursing staff was also infected, and nobody had diarrhea. Do you regard that as a human experiment? I do not know. But I cannot believe that that salmonella was virulent. A susceptible age group was infected, large numbers were infected, the environment was heavily infected, everything was perfect for human disease, but there were no sick patients.

DR. AGER: I should like to comment on the question of age specificity of attack, a question which has intrigued me for some time. I think it is really a question of severity. In very small children, we see high fever, rapid dehydration, febrile convulsions, and other problems which help to bring them to the attention of physicians and, therefore, to diagnosis. I have investigated many cases of salmonellosis in homes in which I have been able to obtain positive cultures from three or four small children who had no symptoms of any kind. Thus, I do not think that age per se determines whether or not a person will develop some kind of illness when he is infected with a salmonella. What is much more important is that if a child develops salmonellosis, he may become ill enough to see a physician and have a diagnosis made. I really think that the question of age specificity as we see it in reports — age-specific incidence being so much higher in the very young — is an artifact based on parental and medical concern and other factors of this sort.

DR. STEELE: I would like to call attention to the S. panama outbreak of 1943 and 1944 in Michigan, where the mortality rate was extremely high in infants during the first week after birth and decreased among the older infants. The younger the children were, the greater the hazard to their life. There were many nurse carriers who were working in the hospital for a long period of time; few of them actually showed any symptoms.

DR. AGER: I agree with that. However, I do not agree with the suggestion that older persons do not have symptoms while children do. Rather, it seems to me that the child tends to respond in a more frightening manner. This is not a matter of an asymptomatic infection as opposed to symptomatic, but rather, a matter of severely symptomatic as opposed to mildly symptomatic.

DR. STEELE: I would say those nurses and nurses aids contributed to the dissemination of the organism, but never developed symptoms This is very important.

DR. AGER: Small children may also remain asymptomatic yet they may have positive cultures. I do not think this is peculiar only to adults.

DR. LANGMUIR: Would you accept dose response by age as a factor of the degree of symptomatology they develop?

DR. AGER: I surely would. I think incubation period and severity are closely related to dose.

DR. NEWELL: This is a question directed to Dr. Edwards.

> QUESTION: Would you discuss the frequency of simultaneous infection with several serotypes?

DR. EDWARDS: There has been a good deal written in the literature about this within recent years. Winkle and Rhode (2) in Germany and Buczowski (3) in Poland have mentioned it, as well as several others. Almost all of them feel that the simultaneous isolation of several serotypes from a given patient or from a series of patients is due to the presence of multiple serotypes in the foods consumed. This is the general concensus in regard to this question.

DR. NEWELL: There are several questions directed to Dr. Best.

> QUESTION: Is an organized systematic national attempt being made in Canada to identify all facets of the salmonella problem and to correct the problem areas?

DR. BEST: Canada's Food and Drug Laboratories have worked closely with food manufacturers. However, there has been a lack of tie-in or sharing of information within Canada; and we, and by we I mean the Advisory Committee on Epidemiology, are attempting to improve this situation. The Advisory Committee on Epidemiology is made up of the provincial epidemiologist from each of the 10 provinces, members of our two schools of hygiene, and myself; we are advisory to the Minister through the Dominion Council of Health.

One problem that we thought could be solved almost immediately was this matter of sharing confidential information. For example, when our public health laboratories isolated salmonella from a particular product, how would this information be passed on to the epidemiologists and others who should know? We do not have a suitable system for sharing such information now. However, at a meeting in February 1963, we thought it desirable to establish a confidential circular system which would not only tie-in the provincial epidemiologists but the public health laboratory directors, the Food and Drug people and, also, the Communicable Disease Center here in Atlanta. This has not been instituted yet, but I hope to see it started rather shortly.

In addition, the Advisory Committee on Epidemiology has been asked to explore the gamut of the problem, and we have wondered if we could undertake in Canada something like you are doing here. Anything learned here will certainly be taken back.

> QUESTION: Persons infected with salmonella are frequently told by the physician to have their water supply tested. Animal products and animal contacts are usually ignored. What has been the number of water-borne outbreaks in the past 10 years?

DR. BEST: I do not recall any documented water-borne salmonellosis outbreak in Canada other than typhoid. I think even in the international literature there are relatively few documented outbreaks of water-borne salmonellosis other than typhoid. Certainly, in proportion to the other types of water-borne outbreaks, they are very few.

> QUESTION: In view of the finding that cake mixes have been contaminated for many years, explain why the incidence of S. thompson infection suddenly increased only a few years ago.

DR. THATCHER: My best guess is based on evidence provided by Dr. C. H. Bigland (4) (formerly Veterinary Pathologist, Alberta Department of Agriculture) who showed that S. thompson, formerly a rare organism in Canada, became widely distributed in the late 1950's to Alberta poultry farms, chiefly from one specific hatchery. The poultry farms involved, after receiving many thousands of young birds, obviously allowed this serotype to become well established. It has subsequently been distributed throughout the entire country by the vehicle of processed foods containing egg products.

There are other points which are pertinent and perhaps would answer other questions. We have shown that the evidence points to the infected egg as the principal source of commercial egg contamination in that we have found no statistical correlation between the coliform counts and the presence of salmonellae in commercial egg products. We have argued, therefore, that contamination by fecally contaminated shells was not the major source of infection. Our research veterinarians offer me the opinion — based on work still in progress — that if a chick once exposed to S. thompson should survive, it is likely to become a carrier throughout its laying period and, further, that this organism may not necessarily be detectable in the fecal tract, but is lodged selectively in the ovaries so that the egg may become contaminated before the shell is deposited. In some studies, we have failed to identify salmonella on the surface of some eggs but have identified S. thompson from inside those same eggs.

Salmonella thompson emerged as a common organism in the human population about a year after its emergence as a common finding in poultry. Dr. Bigland gives other evidence to suggest that one might almost predict what will be the common serotypes in the Canadian human population on the basis of the serotypes prevailing in the chicks during the previous year.

> QUESTION: Comment on the effectiveness of the new regulation pertaining to importation and use of salmonella contaminated food.

DR. THATCHER: We believe our regulation has been highly effective. We have limitations, in that policy decrees that the laboratories of the Food and and Drug Directorate do not serve as a control testing agency for all food industries. We, therefore, only test representative samplings of the productivity of a given factory. This is a policy decision. Thus, there is an opportunity for "leakage" because we do not test all production lots. This bears on what I conclude as an absolute need for modern food industry, viz., to have some knowledge of the bacteriology of its own products.

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SOURCES OF SALMONELLA

MODERATOR: ***Dr. Gail M. Dack***

Chain of Infection–Animal to Human

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In most regions of the world, man has a close association with food animals and consequently is exposed constantly to the diseases found in these animals. Salmonellosis is one of these diseases. Even though man may act as a reservoir once he has been infected, the animal source of this group of organisms is still the most important link in the cycle of infection. It is recognized that some salmonella infections of man, such as Salmonella typhi and S. paratyphi B, are transmitted principally through the ingestion of food and water contaminated with infected human feces and urine; and great efforts have been expended in the task of eliminating such accidental contamination. These efforts have resulted in a marked reduction in typhoid fever. In contrast to this decline, however, there has been an increase in the reported incidence of salmonellosis due to other serotypes, probably because we have not been successful in controlling contamination of foods as a result of direct or indirect contact with infected animal tissues and excretions.

As early as 1888, Gärtner recognized that animals were a source of human salmonellosis when he studied an outbreak in 57 persons who became ill after eating meat from a diseased cow (1). Foodborne outbreaks due to salmonellae other than S. typhi have been reported repeatedly since that time. The source of infection has usually been contaminated food of animal origin. Although the public health hazard of salmonellosis in animals was recognized early, it has been the prevailing philosophy that cooking and proper sanitation would control the problem. It is evident that this has not been the case. Had there been a stronger more direct approach to the control of salmonellosis in animals, the present incidence of salmonellosis in man would not exist.

The preoccupation of human medicine with only human medical problems and veterinary medicine with only problems concerning animal health has resulted in poor communication between the two professions on many problems of common interest. Independently advances were made in understanding the clinical nature of salmonellosis in man and in animals and in the treatment of the disease. The unfortunate lack of communication has resulted in a failure to understand the necessity of implementing satisfactory salmonella control programs mutually advantageous to both groups.

Salmon and Smith isolated and identified S. cholera-suis in 1885 and erroneously considered it to be the cause of hog cholera which was later proved to be caused by a virus(2). Salmonella bacteremia in swine is often a secondary infection to cholera and complicates the highly fatal virus disease. The fact that S. cholera-suis is isolated frequently in cases of hog cholera illustrates two phenomena: the ubiquity of salmonellae in host species and the relatively benign nature of the organism in a healthy host until the host's resistance is lowered by a debilitating agent. It is recognized today that infection in man with this S. cholera-suis can cause severe or even fatal disease (3); however, the risk of man contracting infection is unknown.

In 1892, Loeffler isolated S. typhi-murium (Bacillus aertrycke), so named because it caused a disease called "mouse typhoid" (4). It is now isolated two to three times more frequently from animal species and man than any other salmonella type. This frequency of isolation may be attributed to the large number of animal species for which it is pathogenic and may also reflect increased interest in obtaining bacterial isolations to confirm a diagnosis. Epidemics caused by S. typhi-murium are frequently observed in young calves and chicks; mortality rates are high. Human cases are generally related to the contamination of food by any of many animal species.

In 1893, Kilbourne isolated an organism, known today as S. abortus-equi, from the vagina of an aborting mare (5). The symptoms and course of this infection of horses was different from those usually seen in salmonellosis of other species in that it caused abortion following a brief period of septicemia. During the years when horse breeding was conducted on a large scale, this disease was quite prevalent in the United States; but, in recent

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years, it has almost disappeared. Control of this disease in the United States was attributed in part to use of killed organisms as a bacterin; however, there is lack of agreement about the effectiveness of this and other salmonella bacterins. Human disease attributed to *S. abortus-equi* is considered rare.

Rettger in 1909 isolated *S. pullorum* as the cause of "bacillary white diarrhea," now called pullorum disease, in chicks. The seriousness of this problem was such that it threatened the existence of the poultry hatching industry. Through the extensive efforts of veterinary poultry pathologists, specific and effective methods of control were successfully discovered and adopted. Today these control methods have all but eradicated this infection from breeding flocks in the United States. An interesting facet of these methods, which stand out as peculiar to *S. pullorum*, is that the immunological response of fowl to infection allows for a serological method to detect chronically-infected birds. Thus, infected flocks may be found and destroyed. Unfortunately, this immunological response is not a dependable reaction in the majority of other animals infected with salmonellae, and serological testing cannot be relied upon as an effective method of detection and control.

We have not arrived at the point where it is possible to accurately determine the prevalence of salmonellosis in any species of animals by State or region of the country, nor is it possible to accurately estimate the number of human infections. It has been possible, however, to observe the trends in disease prevalence by species through laboratory reports of isolations received for serotyping. This information supports the opinion that the amount of animal infections encountered directly affects the amount of human disease experienced.

On the basis of the isolations obtained by species, it is possible to obtain an erroneous opinion of the importance of one animal host over another as being the most significant reservoir of salmonellae. The following table presents isolations from domestic animals, reported to the Communicable Disease Center for 1963. According to these figures poultry were the largest single reservoir of salmonellae; 53.4 percent of total non-human isolations were reported in this species. In no other domestic animal, however, is such a concerted attempt made to isolate salmonellae. It may be that if more isolations were attempted from swine or cattle, one of these species would lead the list. Isolations of salmonellae from cattle are common, particularly from young calves, which are a significant source of contaminated meat. From the standpoint of human disease, however, poultry and eggs are incriminated most frequently as the source of infection; and, therefore, attention toward control is logically focused on poultry.

Table 1.–*Total salmonella isolations from common domestic animals reported to Salmonella Surveillance Unit - 1963*[1]

[Total salmonella isolates for 1963, non-human sources - 5,196.]

Species	Total	Percent Non-human
Turkeys	1,430	27.5
Chickens	1,345	25.9
Cattle	580	11.2
Swine	557	10.7
Dogs	70	1.4
Total	3,982	76.7

[1] Source: CDC Salmonella Surveillance Reports for 1963.

There is a high prevalence of salmonella infection in a variety of pet animals including dogs, cats, birds, and reptiles. Little is known about the potential spread of salmonellosis from animals to stockmen or pet owners by contact. Occasional cases in man have been reported in homes where the same serotype has been isolated from the pet dog. Within the past year, there have been reports of three infected families which had pet turtles that yielded the same serotypes. Limited studies on pet turtles have revealed that many of them harbor salmonellae (6). This is not so strange when it is known that at least one commercial supplier of turtles fed the breeding stock rendered meat scraps and raw chicken offal; both types of feed notoriously are contaminated with salmonellae. Pet Easter chicks and ducks have also served as sources of human infection. The intimate contact between a child and these pets satisfactorily explains how this may occur. Fecal material from a caged parakeet was the likely source of a case in a young child (7).

The economic toll of animal salmonellosis is based on losses caused by acute disease which is usually more severe in young animals. Carrier animals and asymptomatic infection of the more resistant adults maintain the infection cycle.

The importance of animal salmonellosis is not accepted as a serious threat to animal industries at the production level even though it may be possible to isolate the organisms from a high percentage of apparently healthy adult animals. These carrier animals serve as sources of infection to other animals and to man. Animals in commerce are held in saleyards and stockyards where they are sorted into new groups for resale to farmers for finishing or to meat packers. This process of movement provides an excellent opportunity for exposure of susceptible animal populations to infected animals and environments. The contamination of holding pens and the build-up of infection in animals has been demonstrated in studies by Galton *et al.* (8), Shotts *et al.* (9), and others. Infected animals become the source of slaughter plant contamination, which has been difficult to control.

There is also variability in pathogenicity of types and strains of salmonellae, which determine the clinical disease in the animal host. Infections with the less pathogenic types are often not apparent; yet, under special situations, such as high dosage and lowered host susceptibility, they do produce disease.

In summary, the basic problem is the control of human salmonellosis. It would not be a problem were it not for the prevalence of infection in animals and the inherent weaknesses in processing methods which allow contamination to continue to occur during preparation and handling of foods derived from animals. The cycle of salmonellosis is allowed to continue in animal populations. Healthy carriers infect new susceptibles, or the organism is introduced into the environment by contaminated animal feeds.

To significantly reduce the human incidence of salmonellosis, it is imperative that attempts be made to control the infection in fowls and other animals. This will require advances in techniques of animal husbandry and development and use of methods that will reduce animal infections from such common sources as contaminated feeds.

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Salmonella in Animal Feeds and Feed Ingredients

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Salmonella infections of livestock and poultry continue to be important problems in the United States as well as other areas of the world. In recent years, a great deal of evidence has been accumulated which shows that feed ingredients are frequently contaminated with salmonella organisms. However, information is very limited concerning the relationship of contaminated feedstuffs to active and inapparent infections in livestock and poultry. The primary reason for the scarcity of information is the time lag between reporting and confirmation of the diagnosis and field investigations of the paratyphoid outbreaks in animals. In most States, field investigations of salmonella infections other than pullorum disease and fowl typhoid are done on a very limited basis.

During the past 30 years in Minnesota, there has been considerable interest in the salmonella problems in livestock and poultry, particularly in turkeys. Approximately 60 serotypes of salmonella have been isolated from groups of young turkeys submitted from 1,379 field outbreaks. About 30 percent of the isolations have been Salmonella typhi-murium. During the period from July 1, 1962, to December 31, 1963, isolations from adult turkeys that have reacted to groups B and D antigens have indicated that other types are emerging in significant numbers. In 204 flocks found infected, 22 serotypes were isolated: S. heidelberg was isolated from 47 flocks; S. typhimurium, from 33; S. saint-paul, from 28; and S. chester, from 22. In this period, 18 serotypes were isolated from young poults submitted to the diagnostic laboratory, and many of the same serotypes were encountered. Unfortunately, epidemiological studies were not done in depth to determine if contaminated feedstuffs were responsible for the introduction of the salmonellae. Examination of fecal samples from patients (horses, cattle, and dogs) presented to the veterinary clinic during this same period indicated the presence of salmonella in 24 of 383 dogs examined, or 6.2 percent; in 18 of 247 cattle examined, or 7.2 percent; and in 48 of 496 horses examined, or 9.6 percent. Unfortunately, no attempts were made to determine if contaminated feedstuffs played any role as the source of the salmonellae.

LITERATURE REVIEW

It is not the intent of this paper to make an extensive review of the literature but to point out a few of the papers that have presented information correlating contaminated feedstuffs with salmonella outbreaks in animals and poultry.

The presence of salmonella organisms in feedstuffs, expecially in commercially prepared poultry feed, was indicated first by Erwin (1) in this country. He examined 206 bags of poultry feed comprising products of 9 commercial companies over a period of 18 months. Erwin was able to show that salmonella-like colonies were found in 77 samples, and S. oranienburg was isolated in three instances from mash granules and concentrates. Proteus mirabilis was recovered once, and the remaining 73 recoveries were identified as Paracolobactrum spp. There was no attempt to relate the contaminated feed with infections in poultry.

Boyer et al. (2), described an outbreak of S. thomasville which was associated with consumption of contaminated feed. There was clear cut proof that the S. thomasville, which caused 34 percent mortality in the poults, came from the feed eaten by the poults. The authors pointed out that this was the first time that this serotype had been isolated from poults in the area and also from an unopened sack of feed that was being used to feed the poults. Other salmonella serotypes were isolated from material cooked in rendering plants and stored in open piles on the floors of the warehouses. There was ample opportunity for the meat scraps to become contaminated. The authors also observed the presence of salmonellae in the feces of young poults without morbidity. Circumstantial evidence indicates that the feed was the source of the salmonellae.

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Watkins *et al.* (3), examined 200 samples of processed animal by-products used in feeds, and 37 salmonella isolations involving 28 serotypes were made from the poultry feeds and animal by-products. Although these studies showed the presence of salmonella organisms in animal by-products, there was no indication that such feeds were sources of transmission of infection in poultry and livestock.

Grumbles and Flowers (4) reported the isolation of 6 serotypes in 5.1 percent of 135 samples of soybean and cottonseed meal tested. These studies pointed out that vegetable proteins can also be sources of salmonellae, but again there was no indication by the authors that such products were involved in outbreaks of salmonellosis in animals.

Pomeroy and Grady (5) during a study in Minnesota examined animal protein supplements and found them to be contaminated with salmonella organisms. They examined 666 samples, mostly animal protein supplements, and found 156 or 23 percent of the samples positive for salmonellae. In the by-products, 25 percent of the samples were positive; and, of the complete feeds, 50 percent were positive. The authors were able to trace the infection in poults in one study to feed contaminated with *S. senftenberg*. The steamed bone meal used in the complete feed was found contaminated with the same serotype that was isolated from the poults.

Pomeroy and Grady (6) found a correlation between the salmonella organisms isolated from poultry and livestock submitted to diagnostic laboratories and the ones isolated from animal by-products. Forty-three serotypes were isolated from feed ingredients; and, over a 25-year period, approximately 60 serotypes have been isolated from livestock and poultry.

Boyer *et al.* (7), in another study showed a correlation between salmonellosis in turkey poults and chicks and contaminated feed. The authors found agreement in salmonella serotypes isolated from poults and the feed fed to these poults. In one case, *S. thomasville* was isolated from the livers and intestinal tracts of the poults and from an unopened sack of the same delivery lot as that fed to these poults. In a second case, four serotypes were isolated from the poults, and two of the types were recovered from the feed. In another case, the authors found an agreement between salmonellae isolated from the chicks and the feed being used. *Salmonella infantis*, *S. anatum*, and *S. derby* were isolated from the ceca; and samples of feed taken from unopened bags at the farm yielded *S. california*, *S. infantis*, and *S. schwarzengrund*. The authors also mentioned two other instances where different serotypes were isolated from the poults and the feed.

Morehouse *et al.* (8), reported on a comprehensive survey conducted by the Animal Disease Eradication Division of the U. S. Department of Agriculture on the problem of feedstuffs being contaminated with salmonellae and other pathogenic organisms. These studies showed the results received from 31 States, where isolation attempts were made from 5,172 samples and 718 contained salmonellae. There were 59 serotypes isolated from 14 different animal by-products and from complete feeds. The serotypes most frequently recovered included *S. montevideo*, *S. senftenberg*, *S. typhi-murium*, *S. cubana*, *S. infantis* and *S. oranienburg*. These authors emphasized the threat posed by these potential pathogens in animal by-products, and recommended further study to obtain definitive evidence that contaminated animal by-products in rations are responsible for specific outbreaks of salmonellosis.

Fromme and Gaase (9), described the potential danger and risk of infection due to salmonella contaminated feed and fertilizers which were involved in outbreaks associated with such products. They stated that *S. indiana* which was not known in Germany was isolated from chicken feed containing imported fish meal, and some of the chickens which had received this feed showed agglutination titers of 1:200 against this organism.

Hirsch and Sapiro-Hirsch (10) reported an outbreak of food poisoning in Israel due to *S. hadar*; the suspected vehicle of infection was chicken livers. In order to trace the infection in the fowl, chicken meal and bone meal were examined and found to be contaminated with the same serotype which was isolated from patients with gastroenteritis.

Rutqvist and Thal (11) during the 1-year period 1956-1957 bacteriologically examined 9,377 samples for salmonella. These authors reported the isolation of 308 salmonella strains representing 15 different serotypes from various animal species and feed samples largely obtained from farms suspected of salmonellosis. Another 161 salmonella strains consisting of 32 different serotypes were isolated from imported meat meal; and 230 strains involving 18 serotypes were isolated from foodstuffs, such as beef, veal, and horse meat. Rutqvist and Thal believed that many instances of salmonella infections in poultry flocks in Norway probably arose through ingestion of contaminated feed. These workers also showed an association between outbreaks of salmonellosis in human beings and salmonella infection of poultry. The same salmonella serotype, *S. montevideo*, was isolated from the poultry and infected human beings.

Muller (12) showed that in Denmark, salmonella types which were found occasionally are now found with increasing frequency from calves and poultry. He believed this was related to the importation into Denmark of meat and bone meal, blood meal, bone and fish meal which proved to be contaminated with many types of salmonella bacteria.

Griffin (13) described the recovery of *S. newport* and other organisms from dog food that was used in mouse breeding colonies that were experiencing outbreaks of S. newport. These findings pointed out the difficulty of maintaining colonies of mice and other laboratory animals free of salmonella infections.

Gray et al., in Australia (14) described an outbreak of bovine salmonellosis which was traced to proprietory cow meal and bone meal salt lick. The contaminated ingredient was the bone meal. The distribution of salmonella species in the bone meal was the same as that in the cow meal sample. Examination of the total output of the largest bone meal manufacturer was shown to be uniformly contaminated with salmonellae.

McClarin et al., in Ireland (15) described an outbreak of salmonellosis in pigs, which was due to salmonella-contaminated pig meal. The mills preparing the foodstuffs were visited, and some of the ingredients which were used in the final product were examined. The same salmonella types were found in the pig meal at the mill and also in the fish meal and bone meal used in the complete feed. The authors were of the opinion that some salmonella types pass from feedstuffs through pigs to human food and cause human infections. They also emphasized that the organisms will continue to be passed to human beings unless it is recognized that the only effective way to break the chain of infection is by attempting to break the animal cycle. The authors suggested that this is possible by bacteriological control of animal feedstuffs.

Knox et al., in England (16) described a milk-borne outbreak of food poisoning due to S. heidelberg. The source of infection was a cow suffering from mastitis, and S. heidelberg was isolated from the udder of the cow. In an effort to trace how the organism reached the cow, feedstuffs which were supplied to the farm were examined and were found to be contaminated with several salmonella serotypes. S. heidelberg was isolated from the meat and bone meal at the suppliers factory. This serotype was uncommon in animal feedstuffs and the isolation of the organism from the cow and from meat and bone meal suggested that the two might be associated.

Rohde and Bischoff (17) bacteriologically examined fish meal which they stated was fed very extensively to pigs in Germany. They also emphasized that a growing list of human infections with previously rare salmonellae serotypes have been reported and that this list was strikingly similar to species recovered from the fish meal.

Siddiqui (18) described the isolation of S. anatum from poults which received contaminated feed during an outbreak of salmonellosis on a turkey farm in Minnnesota. The mortality experienced in poults with this type was approximately 33 percent. The infection in the poults was traced to the turkey feed contaminated with S. anatum. The same serotype was isolated from the feed and the livers and intestinal content of the poults.

Pomeroy and Siddiqui (19) in an effort to trace the source of an unexplained outbreak of salmonellosis in turkeys examined various samples—including dead poults, feed, litter, and mice droppings—collected from a farm. S. infantis and S. derby were isolated from the litter samples and from the poults. The mortality experienced in the poults with these two serotypes was around 10 percent. The mice droppings were positive for salmonella and the serotype was identified as S. infantis. There was a strong indication that the infection originated from the contamination of feedstuffs with mice droppings because samples from unopened sacks of feed were negative.

Galton et al. (20), examined a total of 159 samples of dehydrated dog foods. Salmonellae were isolated from 26 (26.5%) of 98 samples of dog meal, but none were found in 54 samples of pressed foods or 7 samples of dog "candy." During this study, 17 serotypes were isolated. All of these types except S. lexington were obtained from dogs in Florida, and 15 were isolated from human beings.

Shotts et al. (21), reported the recovery of salmonellae from 23 percent of the animal feeds and from approximately 50 percent of animal feed ingredients and rendered material. In these studies, a total of 38 salmonella serotypes were isolated and identified. Nearly one-half of the serotypes were recovered more than once during the investigation. In seven instances, the same serotype was found to be present in feeds, swine, and the slaughter plant environment. Nine serotypes were isolated from the swine and from sausages and not from the feeds, while 21 serotypes which were not found in animals or sausages were isolated from the feeds. From these studies, it is obvious that many animal protein supplements are contaminated with salmonella and offer a source of infection. These infected animals may eventually find their way to slaughter. During the slaughter, the carcasses frequently become contaminated with salmonella. The foods prepared from these carcasses are potential sources of human infection.

Burr and Helmboldt (22) examined 436 animal by-products and feed-ingredient samples for the presence of salmonella. Fifty-six (12.8%) samples were found to be contaminated with salmonellae, and the different serotypes were identified. In these studies, S. derby was isolated two times, once from a sample of poultry by-products and the other time from meat scraps. These authors were of the opinion that infection of flocks with salmonellae, as a result of feeding of salmonella contaminated feed, could cause non-specific agglutination reactions when the flocks are tested in the pullorum-typhoid testing program. In supporting this view, the authors cited the trouble encountered in 1955-56 in the Connecticut pullorum testing program in which flocks had positive pullorum agglutination tests as a result of infection with S. heidelberg. The sources of these infections were undetermined.

SUMMARY

1. The numerous reports in the literature have clearly shown that feed ingredients which are used to formulate complete feeds for livestock and poultry are frequently contaminated with salmonella organisms.
2. Recontamination of the ingredients—particularly animal, poultry, and fish by-products—after their processing is considered the chief factor resulting in the presence of salmonellae in these products.
3. Definitive evidence that contaminated feeds have been sources of salmonella outbreaks in animals and poultry is well documented, but a limited number of reports are available. Unfortunately, extensive epidemiological studies have not been made on field outbreaks of salmonellosis in animals and poultry with the exception of pullorum disease and fowl typhoid.
4. Because of the wide host range of salmonella and the ubiquitous nature of the organism, consideration should be given to the development of sanitary methods that will assure the final complete feed to be free from pathogens. This may require the "pasteurization" of the feed in the final stages of manufacture.

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Salmonella in Poultry and Eggs

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The presence of salmonella organisms in poultry and eggs has been recognized for the past six decades. Since the early part of this period, two serotypes, *Salmonella pullorum* and *S. gallinarum,* have been of great economic significance to the chicken and turkey industries. In more recent years, the incidence of the diseases caused by these organisms, as well as their economic threat to the industry, has been reduced greatly. During the past three decades, other salmonella infections have come into prominence because of their occurrence in poultry and eggs (1-6). Some serotypes have been identified more frequently than others; and, up to the present, a total of 108 specific types have been isolated from chickens and turkeys (1, 7). The economic losses caused by the various types of salmonella in poultry flocks, especially in young birds, is of concern to the poultry industry (1,3-6). Of even greater significance is the fact that a number of these serotypes, transmitted in various ways, have been responsible for numerous sporadic cases and epidemic incidents of human salmonellosis (2, 8-11).

The complexity of avian salmonellosis should be recognized in view of the fact that many serotypes, that have been identified, may be pathogenic for a broad spectrum of hosts, including man (2). Their geographical distribution is world-wide; but, in many countries, including the United States, reliable information is lacking as to their frequency of occurrence in man and animal.

The purpose of this paper is to assess the prevalence of salmonella infections in poultry and eggs, and its significance to public health. Many excellent reviews, research findings, and epidemiological reports have been documented by workers in this country and by our colleagues elsewhere (2, 4). It is gratifying that an increasing interest in this subject is evident in persons concerned with human and animal health. Persons engaged in the study and control of poultry diseases are showing a greater awareness of the economic threat of salmonella infections and of their significance to public health (12-14).

SALMONELLA INFECTIONS IN DOMESTIC FOWL

Williams (1), in a comprehensive and excellent treatise on avian salmonellosis, reported that 108 serotypes have been isolated from chickens and turkeys in the United States. Additional types, isolated from fowl, have been reported in other parts of the world. Salmonella infections are frequently encountered in other avian species—especially ducks, pigeons, and canaries.

Two serotypes, *S. pullorum* and *S. gallinarum,* have been recognized in the chicken for more than a half century. The diseases produced by these organisms have taken a great toll from the poultry and turkey industries. Both diseases are egg-borne; but fortunately their host range is largely confined to chickens and turkeys, although a number of serious cases of salmonellosis due to *S. pullorum* have been reported in man (15). However, through effective testing and eradication procedures, the incidence of these two diseases has been reduced greatly among chickens and turkeys. Both diseases appear amenable to eradication if our present knowledge concerning these infections could be applied effectively through cooperative effort by those concerned (16) (Table 1).

Other salmonella types associated with poultry may produce signs and lesions that are characteristic of salmonellosis. Since this aspect of the problem has been described elsewhere by several investigators, it will not be repeated in this paper (1, 3, 4, 17). Clinical salmonellosis is primarily seen in the young fowl, although it may occur rarely in mature birds. The pattern of an outbreak may be influenced by the serotype involved, its virulence, the resistance of the host, and the environmental conditions to which the young and older fowl are subjected. Mortality in young chicks, poults, and ducklings may approach 25 to 30 percent during the first 2 or 3 weeks of age. The source of the infection can be traced usually to the breeding flock in which hens with either ovarian or an intestinal infection may contaminate the eggs. A salient feature of salmonellosis is that young and mature birds surviving an experience with these organisms may become carriers of the infection hence perpetuating the disease cycle. This may involve single and multiple serotypes in successive generations of stock (2, 3, 17).

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All diagnoses of salmonelloses should be based on the isolation and identification of the serotype. To isolate these organisms one should select cultures from many tissue sites. In acute cases, the organism may be recovered from many sites, including the brain and bone marrow, while in carriers the intestinal tract is more apt to yield the organism. Some of the serotypes have been isolated from the female reproductive tract. The rate of recovery from the ovary and oviduct is very low, but it should be recognized that the organism may be transmitted from generation to generation by its presence in the interior of the egg (18-20).

Table 1.–*Pullorum testing data submitted by states and Canadian provinces in the northeastern conference on avian diseases*

State	Year	Number of birds tested	Percentage positive
Connecticut	1925	20,743	2.40
	1963	470,191	0.00
Delaware	1925	4,300	5.70
	1963	522,260	0.00
Maine	1921	2,730	22.30
	1963	1,558,928	0.00
Maryland	1927	3,725	21.00
	1963	337,195	0.00
Massachusetts	1921	24,718	12.50
	1963	589,005	0.00
New Hampshire	1926	35,237	2.50
	1963	442,676	0.00
New Jersey	1926	52,611	7.86
	1963	395,574	0.00
New York	1926	59,576	6.20
	1963	478,827	0.00
North Carolina	1932	64,702	4.02
	1963	3,788,963	0.0006
Nova Scotia	1929	2,041	7.00
	1963	68,145	0.00
Ontario	1928	15,000	8.00
	1963	1,233,128	0.003
Pennsylvania	1924	2,077	15.00
	1963	1,344,923	0.0008
Rhode Island	1925	8,175	6.97
	1963	54,642	0.00
Vermont	1928	8,555	7.40
	1963	101,216	0.00
Virginia	1925	13,000	20.00
	1963	1,051,514	0.006
West Virginia	1928	9,005	6.00
	1963	49,914	0.00

SALMONELLA IN EGGS

Transient or chronic carriers that harbor salmonella in the intestinal tract may contaminate the exterior of the egg as it is laid or when it comes in contact with contaminated nesting material or litter (18, 19, 21-23). It has been demonstrated conclusively that surface contamination with salmonella may result in the organism's penetrating the shell and the interior of the egg. Cantor and McFarlane (21) were successful in isolating salmonella from shell scrapings and egg meats from chicken eggs obtained from commercial channels. In no instances were organisms isolated from both the shell scrapings and egg meats from the same eggs. S. pullorum was isolated only from the egg meats. The positive isolations were obtained more frequently from eggs of the four lower United States grades than from the four higher grades. It was emphasized that egg processors will have a more serious microbial problem as long as eggs of the inferior grades are considered eligible for edible grades. Stokes et al. (24), have demonstrated that salmonella organisms can penetrate the egg shell when the surface of the egg is contaminated with the organism. This applies to both the motile and non-motile serotypes of salmonella. The degree of contamination and the temperature at which the eggs were stored influenced the amount of growth within the eggs.

Our knowledge of the presence of salmonella in eggs emphasizes that sanitation may play a very significant role in reducing the spread of infection by the egg. Superior sanitary management in the handling of eggs from the farm level to the ultimate consumer should be stressed to reduce the occurrence of salmonella in eggs or egg products. The presence of the organism either on the egg's surface or internally appears to be an important mode of transmission and perpetuation of salmonellosis in chickens, turkeys, and ducks (1, 6, 18, 25).

Various salmonella serotypes appear to survive for long periods under certain types of environment. S. pullorum has remained viable on a dry cloth, maintained at room temperature for at least 7 years and 8 months (26). Other investigators (27-30) have reported that various salmonellae, especially S. typhi-murium, may remain viable under different environmental conditions for prolonged periods. In one instance, S. typhi-murium remained viable within the egg for 13 months when maintained at incubator temperature (27). On the other hand, Lancaster and Crabb (32) state that S. typhimurium and S. thompson rapidly lose their viability on the shell of whole chicken eggs maintained at incubator temperature. At room temperature these organisms survived for approximately 21 days on clean eggs and for a longer period on artifically dirty eggs. Increased humidity prolonged the viability of the organisms. Belcian and Schlosser (33) reported that the size of the egg mass and the degree of contamination are two important factors that influence the destruction of the organism in baking.

SALMONELLA IN EGG PRODUCTS

The use of contaminated shell eggs has resulted in salmonella contamination processed eggs and egg products (12, 20, 23). This has resulted in widespread contamination of processed egg products, as well as contamination of equipment and facilities in the processing plants.

Solowey *et al.* (34), in 1947 reported an extensive investigation which revealed that salmonellae were found in 35 percent of 5,198 samples of spray dried whole egg powder obtained from 100 dehydration plants. The incidence of salmonella in samples examined from individual plants ranged from 0 to 71 percent. Fifty serotypes were identified of which five (*S. pullorum*, *S. oranienburg*, *S. montevideo*, *S. tennessee*, and *S. anatum*) made up 73 percent. *Salmonella pullorum* represented 28 percent of the isolations. Felsenfeld *et al.* (35), found 3 percent of 100 samples of dried egg powder contaminated with *S. derby*, *S. montevideo*, and *S. panama*.

In 1961, North (36) reported that the majority of dried albumen products from most egg processing plants were free of salmonella. The high incidence in certain plants could be attributed to contamination being carried over from one batch of albumen to the next. Attention is called to improvements in methodology in the processing and checking of the products. In 1963, Kampelmacher (9) reported that a number of investigators in Germany had made extensive surveys of imported egg products and recovered salmonella, representing many serotypes, from 2 to 5 percent of the samples tested. Frozen eggs and egg powders yeilded the highest percentages of salmonella. Imports from the United States were the highest in salmonella isolates. In Holland, 4.5 percent of 3,000 lots of egg products examined from 1953-59 were found positive; *S. typhi-murium* occurred most frequently. However, in 400 lots of duck egg products, 19 percent were positive; *S. typhi-murium* and *S. enteritidis* revealed the highest incidence. Hobbs (20) reported the presence of salmonella in various types of egg products received from domestic and foreign sources. Among the samples examined during 1961-62, salmonellae were identified in 11 and 20 percent respectively. *Salmonella paratyphi* B, *S. typhi-murium*, *S. thompson*, *S. montevideo*, *S. potsdam*, and *S. tennessee* appeared most frequently. At the present time, approximately 100 official plants that produce a variety of egg products exist in the United States (37). Twenty-six of the larger plants are equipped with facilities for pasteurizing egg products other than albumen. Plants pasteurize their products at 141° to 143°F. for 3-1/2 minutes. Some plants have their own laboratory and technicians who continuously make bacteriological examinations. The United States Department of Agriculture has four official laboratories for test purposes. However, bacteriological examination of a product is not a requirement of the Federal regulations. The British workers recommend that for adequate pasteurization of whole egg products a temperature of 148°F. for the minimum holding time of 2-1/2 minutes should be used (38).

Since the incidence of salmonella organisms has been high among certain samples of egg products, it would seem logical and expedient to reassess this food processing program with the hope of further reducing the degree of infection among these different food commodities.

SALMONELLA IN DRESSED POULTRY

Modern poultry dressing and processing had made great progress in furnishing the consumer with high quality poultry meat. Through improved sanitation and hygiene practices in the plants, together with competent Federal and State inspection services, the objective is to produce a wholesome dressed bird.

Bacteriological surveys made of slaughtered poultry at various stages of the processing in plants have revealed that various types of salmonella could be recovered. Brobst *et al.* (39), found five serotypes in a poultry processing plant not subject to Federal inspection. The water bath used after defeathering yielded the greatest number of positive isolations. In another plant, Felsenfeld *et al.* (35), found 14 percent of the carcasses infected that had been federally inspected and passed; whereas, 26 percent of the non-inspected carcasses were infected. Poultry dressed in the home and non-inspected revealed a high incidence of salmonella representing 13 different serotypes.

Sadler *et al.* (40), reported on a 20-month survey conducted in California of processed fryer chickens, hens, and turkeys obtained from dressing plants. Forty-four flocks were sampled of which 21 yielded salmonella. The rate of infection was higher among the turkeys (37 percent) and fryers (18 percent) than among the hens (10 percent). *Salmonella typhi-murium* was isolated most frequently from the turkeys and hens. *Salmonella infantis* and *S. bredeney* were more prominent among the fryers. Other serotypes isolated included *S. heidelberg*, *S. give*, *S. san-diego*, *S. javiana*, *S. panama*, and *S. worthington*.

Following an extensive survey, Galton *et al.* (41), reported on the presence of salmonella in poultry in Florida. Observations made in three poultry processing plants revealed that salmonella-infected birds were submitted for slaughter and that the salmonella could be widely disseminated in a plant during the processing. Samples obtained from tubs holding iced birds ready for market were found to be 11 percent positive. The authors stated that there is no clear evidence that salmonellae persist or multiply in a contaminated plant. Among 1,244 cultures taken, 16 percent were positive. The highest percentage of positive cultures originated from the edible viscera and the table on which edible viscera were wrapped. A total of 24 serotypes was identified.

Dixon and Pooley (42) found the water in chilling vats of a poultry processing plant to be highly contaminated with salmonella. Swabs taken from the gloves of employees yielded a serotype that was not detected in specimens collected from other sources. Also, among specimens collected on 23 different days, only those collected on 13 of the days yielded salmonella. Schneider and Gunderson (43) examined 1,014 eviscerated chickens of which 4.4 percent yielded salmonella. Carcasses cultured immediately after evisceration showed less contamination with S. pullorum than those cultured just prior to packaging which was attributed to spread by the process of removing infected ovaries.

Kampelmacher (23) cites the findings of numerous workers in the European countries who reported the isolation of salmonella from dressed poultry suggesting that such contamination is of concern to all countries employing the more modern processing procedures.

HUMAN SALMONELLOSIS OF AVIAN ORIGIN

Sporadic cases and epidemic outbreaks of human salmonellosis have been reported more frequently in the last few years. Human salmonella isolates reported to the Communicable Disease Center from September 1, 1962, to December 31, 1963, revealed the following types to be among the first seven in prominence: S. typhi-murium, S. heidelberg, S. derby, S. newport, S. infantis, S. typhi, and S. enteritidis (44). For a somewhat similar period (January 1962 to July 1963), the National Animal Disease Laboratory found the following as the more frequent serotypes of chicken and turkey origin: S. typhi-murium, S. heidelberg, S. saint-paul, S. chester, S. infantis, S. montevideo, and S. san-diego (45). It is gratifying that the staff at the Communicable Disease Center, Federal and State public health workers, physicians, and veterinarians have demonstrated an increasing interest in human and animal salmonellosis.

The literature reveals that a number of human outbreaks of salmonellosis have been traced to avian sources. Among the 108 salmonellae identified in avian species, certain serotypes have been incriminated more frequently than others for human outbreaks. Many of these cases are well-documented and will not be discussed in this paper since they may be reviewed by other speakers on the program (8-11, 44-50).

In order to cope with salmonellosis in animals and birds, it is necessary to assure that animal feeds are free of salmonella in order to prevent reinfection once a herd or flock has been declared free of these organisms. The incidents of salmonella infection in animals, including the avian species, should be made a reportable disease as has been recommended by Edwards (51). Isolates from all animal and avian sources should be serotyped and adequate histories should be provided to State and Federal typing agencies. A central salmonella surveillance agency should be established for incidents of salmonella infection in animals and poultry. All incidents of salmonellosis in animals and poultry should be subjected to effective control and eradication measures. All poultry and egg processing plants should strive to develop and employ methods that will assure a wholesome product for human consumption. With our present knowledge of food hygiene, it is reasonable and logical to expect that all processors of poultry and eggs make a real effort to attain this goal.

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Salmonella in Foods

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In recent years, certain common foods and food ingredients used by manufacturers, canteens, and housewives have been found to be consistently contaminated with salmonellae; Salmonella typhi-murium, S. paratyphi B, and other serotypes – some commonly and some rarely found in human salmonellosis – have been isolated.

Egg products, meat, and coconut have all been related to widespread and scattered incidents of gastroenteritis and enteric fever due to paratyphoid bacilli and other salmonellae; and, if the common vehicle had not been suspected, each incident would have remained isolated. Thus, the knowledge that certain types of salmonellae occur in particular foods is helpful, and phage typing has been valuable in finding and linking causative agents such as S. paratyphi B and S. typhi-murium to scattered outbreaks. It may be possible to reduce the salmonella content in foods by an improvement in the hygiene of production. When this is not practicable, some form of treatment may be necessary to rid the product of salmonellae before distribution.

EGG PRODUCTS

Table 1 presents the results of examination of egg products for salmonella in 1961 and 1962. Of 3,422 samples of all egg products examined in 1961, 11 percent contained salmonellae, and of 998 samples examined in 1962, 20 percent were contaminated (1,2).

Table 2 gives figures for 1963. There was a marked improvement in the results from all products; of 2,729 samples, only 6.4 percent were positive. The reduction in the number of samples positive for salmonellae was undoubtedly due to the use of pasteurization in the United Kingdom and in some other countries. The results include all samples whether raw or pasteurized.

Table 3 compares the results of examining raw and pasteurized frozen whole egg from three countries; the fourth sent raw egg only. Of 448 samples of frozen raw whole egg from China, 18.7 percent were contaminated; whereas salmonellae were not isolated from 419 samples of pasteurized egg from the same source.

In tests of frozen liquid egg from the United Kingdom, 21.8 percent of 133 samples of raw egg were found positive; and out of 243 samples of the pasteurized material, none were positive. Out of 161 samples of Australian raw egg examined, 8.7 percent were positive, and approximately 3 percent of 167 samples of egg pasteurized in Australia were positive. Samples from subsequent shipments pasteurized in the same plant were negative for salmonellae. The total reduction of salmonellae in all samples is convincing and justifies the United

Table 1.–*Results from examination of egg products for salmonella, 1961-62*

Egg products examined	Percent samples positive	
	1961	1962
Whole frozen	16	16
Whole dried	12	23
White frozen	6	19
White powder	8	27
White flake	13	19
Yolk dried	5	14
Total samples	[1]11	[2]20

[1]3,442 samples examined. [2]998 samples examined.

Table 2.–*Results from examination of egg products for salmonella, 1963*

Egg products examined	Number of samples examined	Positive for salmonellae	
		Number	Percent
Whole frozen	1,629	141	8.6
Whole dried	325	15	4.6
White frozen	470	1	0.2
White powder	55	6	10.9
White flake	211	13	6.2
Yolk frozen	14	0	....
Yolk dried	25	0	....
Total	2,729	176	6.4

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Kingdom legislation on the pasteurization of liquid whole egg which came into force on January 1, 1964 (3). The condition of 148° F. for 2-1/2 minutes has proved to be practicable, and the alpha-amylase test is in full use (4).

Table 4 gives the results for alpha-amylase tests on pasteurized egg from Australia, China, and the United Kingdom. All samples examined from Australia passed the test, even those which contained S. saint-paul, as shown in Table 3. This suggested postprocessing contamination, because S. saint-paul was not heat-resistant; and it was, therefore, unlikely to have survived even lower temperatures of pasteurization.

The results from China showed that 6.3 percent of 315 samples of frozen egg pasteurized in the United Kingdom and 65.4 percent of 104 samples from egg pasteurized in China failed the test, although salmonellae were not found. The results suggested that the Chinese material was pasteurized at a lower temperature perhaps for a longer time. Of 243 samples pasteurized in the United Kingdom, 16.8 percent failed the test but salmonellae were not found. The pasteurization of liquid whole egg at 148° F. for 2-1/2 minutes has proved to be satisfactory for the destruction of alpha-amylase and of heat-resistant strains of salmonellae (5). It also gives a satisfactory product for baking (6); lower temperatures for a longer time may destroy alpha-amylase, but the effect of this treatment on S. senftenberg 775 W and on the baking properties is doubtful.

Some of the outbreaks of paratyphoid fever and salmonellosis in which the causative organism originated from egg products have been described in the literature (7-11).

FROZEN-PACKED BONELESS MEAT

The amount of packed frozen boneless meat imported into the United Kingdom is small compared with that of imported and home-produced carcass meat. Yet imported boneless beef, veal, and mutton are used predominantly in manufactured meat products such as sausages and pies. Parry (12) reports that 9,000 million raw meat sausages are sold annually in the United Kingdom, and according to the Food Standards Committee (13) they account for at least 10 percent of the total weight of meat consumed. This meat is used also by canteens, in hospitals,

Table 3.–*Salmonellae from frozen whole egg, 1963*

Country of origin	Number of samples examined	Raw			Pasteurized		
		Number of samples examined	Positive for salmonellae		Number of samples examined	Positive for salmonellae	
			Number	Percent		Number	Percent
Australia	328	161	14	8.7	156(A)[1] 11(U.K.)[1]	[2]5 0	3.2 .....
China	867	448	84	18.7	104(C)[1] 315(U.K.)[1]	0 0	....
South Africa	58	58	9	15.5	....	.....	.....
United Kingdom	376	133	29	21.8	243	0	.....
Total	1,629	800	136	17.0	829	5	0.6

[1] Country where pasteurized and examined.

[2] *S. saint-paul.*

Table 4.–*Results of alpha-amylase test for frozen whole egg*

Country of origin	Pasteurized in United Kingdom				Pasteurized in country of origin			
	Number of samples examined	Passed	Failed		Number of samples examined	Passed	Failed	
			No.	%			No.	%
Australia	11	11	0		156	[1]135	0	
China	315	[1]260	[2]20	6.3	104	36	[2]68	65.4
United Kingdom	243	202	[2]41	16.8	....	......	...	....

[1] Not all samples examined. [2] Percent of total.

schools, and factories as a cheap source of protein. It is sold alongside carcass meat and manufactured meat products in many retail shops and both sorts of meat are handled by the same employees. Hobbs and Wilson (14) showed that 10 percent of samples of imported frozen boneless meat were contaminated, compared with 4.3 percent of samples of chilled carcass meat from the same countries.

A small proportion of boneless horsemeat is bought for human consumption; some in canned, but most of the imports are sold by pet shops either raw or mixed with other ingredients as proprietary pet foods. Many animals eat the meat raw, and may become symptomless excretors temporarily; also the kitchens of homes may be contaminated with the salmonellae, spread around by utensils, cloths, and hands (15).

Table 5 gives the percentage of samples of imported boneless frozen meat for human consumption, found to be positive for salmonellae during the period 1961-1963. Veal, beef, and mutton all gave many positive results, but the percentage of contaminated samples varied from country to country and from year to year. The improvement in the results from New Zealand veal in 1963 may be due to the ban on exportation of meat from the worst packing stations after the condemnation of many batches by the Port of London, and to the improved hygiene in many packing stations, as reported by the Meat Research Station in New Zealand (16). Improvements in factory practice also led to better results from Bechuanaland boneless beef which was giving up to 70 percent of positive samples when the first consignments were examined in 1960.

Table 5.–*Salmonellae from frozen boneless raw meat for human consumption, 1961-63*

Type of meat and country of origin	Number of samples examined	Positive for salmonellae	
		Number	Percent
Veal:			
New Zealand	684	147	21.5
Australia	115	3	2.6
Argentina	10	0	0
Total	809	150	18.5
Beef:			
New Zealand	15	0	0
Argentina	1,326	296	22.3
Uruguay	142	28	19.7
South Africa	790	33	4.2
Australia	114	7	6.1
Total	2,387	364	15.2
Mutton:			
New Zealand	121	3	2.5
Australia	43	7	16.3
Argentina	40	10	25.0
Total	204	20	9.8

Most of the results from South American boneless meats, both for human and animal consumption, were poor. Batches of imported boneless meat found to be positive for salmonellae are subject to the Staining and Sterilization Regulations (17) under the Meat Regulations of the Food and Drugs Act. It is required that meat for human consumption which is condemned must be "cooked" before sale for animal food.

Table 6 gives the results for imported boneless horsemeat and offal which comes mainly from South America and Europe. The horsemeat is intended for animal consumption, but it is regarded as fit for human consumption, and therefore it comes under the jurisdiction of the Staining and Sterilization Regulations. The results indicated a high rate of contamination.

Tables 7 and 8 give the results from meat samples obtained from different establishments. They show that all the horsemeat factories and most of the beef factories were packing contaminated meat. Kampe (18) surveyed an Argentine meat establishment which slaughtered horses and packed meat. He described much that was unsatisfactory hygienically. The use of antibiotics in certain establishments may have lowered the level of contamination temporarily but the improvement was not maintained. Huey and Edwards (19) reported an increase in the antibiotic-resistance of S. typhi-murium; Hobbs et al. (20), confirms this observation.

Table 6.–*Salmonellae from frozen boneless raw horsemeat for animal consumption, 1961-63*

Type of meat and country of origin	Number of samples examined	Positive for salmonellae	
		Number	Percent
Horsemeat:			
New Zealand	7	1	14.3
Argentina	4,888	2,135	43.7
Paraguay	291	106	36.4
Bulgaria	83	11	13.2
Rumania	129	23	17.8
Hungary	6	0	0
Poland	11	1	9.1
Total	5,415	2,277	42.0
Horse offal:			
Argentina	486	263	54.1
Paraguay	27	2	7.4
Bulgaria	48	16	33.3
Total	564	281	49.8

Table 7.—*Salmonellae in horsemeat from different meat establishments in Argentina, 1961–63*

Establishment number	Year	Number of samples examined	Positive for salmonellae	
			Number	Percent
33	1962	20	2	10.0
196	1961	66	65	98.5
	1962	62	60	96.8
	1963	7	7	100.0
223	1962	309	192	62.1
	1963	156	105	67.3
331	1963	56	38	67.9
351	1962	9	9	100.0
	1963	55	17	30.9
511	1961	5	0	0
	1963	106	97	91.5
529	1962	3	2	66.6
	1963	45	38	84.4
1344	1961	40	5	12.5
	1962	247	79	31.9
1369	1961	107	87	81.3
	1962	248	204	82.3
	1963	379	227	59.9
1400	1962	18	9	50.0
	1963	60	31	51.7
1411	1961	148	54	36.5
	1962	864	268	31.0
	1963	114	33	28.9
1451	1962	3	3	100.0
1541	1963	1	1	100.0
1821	1963	2	2	100.0
1933	1961	113	67	50.5
	1962	402	56	13.9
	1963	672	154	22.9
1934	1961	88	68	77.3
	1962	31	27	87.1
1965	1963	14	11	78.6
2005	1963	130	3	23.1
2006	1963	20	2	10.0
2009	1963	22	9	40.9

Tables 9 and 10 list the serotypes isolated from various meats. *S. typhi-murium* was the predominant type in boneless veal and beef. The South American meat carried a wide variety of other types also, *S. newport* and *S. anatum* being the most common. Fifteen of the serotypes found in boneless beef from Uruguay and Argentina were also found in horsemeat and offal from the same countries.

The most common serotypes in the horse products were *S. minnesota*, *S. meleagridis*, *S. oranienburg*, *S. anatum*, *S. derby*, and *S. give*, although *S. paratyphi* B, *S. typhi-murium*, and *S. newport*; other serotypes isolated from human incidents were found also. Lists of salmonella serotypes isolated from food and feeding stuffs in relation to the same serotypes isolated from humans and animals have been compiled (21, 22).

The significance of *S. typhi-murium* in boneless meat cannot be assessed in relation to human salmonellosis without the results of phage typing (23).

Table 8.—*Salmonellae in boneless beef from different meat establishments in Argentina, 1961–63*

Establishment number	Year	Number of samples examined	Positive for salmonellae	
			Number	Percent
1	1963	12	0	0
1a	1963	45	1	2.2
2	1963	62	10	16.2
3	1963	25	0	0
7	1963	27	0	0
8	1963	11	5	45.4
12	1961	5	0	0
20	1963	66	17	25.7
25	1963	45	22	48.9
44	1963	121	1	0.8
66	1963	141	32	22.7
1383	1961	25	0	0
	1963	226	88	38.9
1404	1961	10	2	20.0
	1963	152	39	25.6
1464	1961	5	0	0
1930	1963	196	4	2.0
1979	1963	62	25	40.3
1985	1963	90	50	55.5

Man may be infected from many food sources although a common type may be the causative agent (24). Lundbeck, Plazikowski, and Silverstolpe (25) reported a widespread outbreak of food poisoning attributed to *S. typhi-murium* in meat; Kallings, Laurell, and Zetterberg (26) described other incidents due to *S. typhi-murium* in veal, with special reference to phage typing. A phage typing center is essential for any country investigating the source of outbreaks and sporadic cases of salmonellosis and the mode of spread of the causal agents.

RETAIL MEAT AND PET FOOD

Table 11 gives the results of a small comparative survey, carried out in various parts of the United Kingdom, of the salmonella content of meat for human and animal consumption. Samples examined came from retail butchers' shops, pet shops, and knackers yards. An average of 1.5 percent of samples of butchers' meat, 22 percent of samples of meat intended for pets, and 11.2 percent of the samples of knackers meat contained salmonellae. The butchers' meat was mostly English except for the Newcastle samples. The percentage of pet food samples positive for salmonellae was as high as 40 percent in Leeds, although the number of samples was small. Colindale's figure of 26.2 percent was obtained predominantly from samples of imported horsemeat and offal; whereas, the 36.2 percent of positive samples reported from Manchester consisted mostly of butchers' scrap assumed to be home-produced meat. Most of the knackers yard samples had a low rate of contamination; the figure for Newcastle, however, increased the average figure.

Table 9.—*Types of salmonellae and their frequency of isolation from frozen packed boneless raw meat for human consumption, 1961-63*

Serotype	Meat, country, and number of samples positive for salmonellae							
	Veal		Beef			Mutton		
	New Zealand	Australia	Argentina and Uruguay	Bechuanaland and S. Rhodesia	Australia	Argentina	New Zealand	Australia
paratyphi B.	......	......	3	....	......	......	......	......
typhi-murium	140	1	88	13	3	1	1	6
abortus bovis	......	......	......	1	......	......	......	......
anatum	......	1	63	4	1	......	......	......
bovis morbificans	3	......	3	....	......	1	2	1
bredeney	......	1	......	1	......	......	......	......
cerro	......	......	......	1	......	......	......	......
chester	......	1	......	....	1	......	......	......
derby	1	......	7	....	......	......	......	......
gaminara	......	......	1	....	......	......	......	......
gilbert	......	......	......	2	......	......	......	......
give	......	......	12	....	......	3	......	......
halmsted	......	......	1	....	......	......	......	......
hamburg	......	......	......	1	......	......	......	......
heidelberg	2	......	......	....	......	......	......	......
irumu	......	......	......	1	......	......	......	......
kapsted	......	......	......	1	......	......	......	......
loma-linda	......	......	......	5	......	......	......	......
meleagridis	......	......	11	....	......	......	......	......
minnesota	......	......	9	....	......	2	......	......
montevideo	......	......	27	1	......	2	......	......
newport	......	......	80	....	......	1	......	......
oranienburg	......	......	10	....	1	......	......	......
panama	......	......	7	....	......	......	......	......
portbech	......	......	......	1	......	......	......	......
potsdam	......	......	......	1	......	......	......	......
saint-paul	1	......	......	....	......	......	......	......
san-diego	......	......	1	....	......	......	......	......
senftenberg	......	......	5	....	1	......	......	......
Unidentified	......	......	2	....	......	......	......	......
Total samples positive	147	3	324	33	7	10	3	7
Percent samples positive	21.5	2.6	22.1	4.2	6.1	25.0	2.5	16.3
Total samples	684	115	1,468	790	114	40	121	43

It may be inferred from these results that under normal conditions approximately 2 percent of samples of butchers' meat for human consumption in the United Kingdom may contain salmonellae, and that at least one-fifth of pet meat samples are likely to be contaminated in spite of port examinations. More results are needed from knackers' meat, but it is unlikely that the rate of contamination of meat from occasional animals killed because they are sick or injured would be as great as that of boned-out meat stripped from hundreds of carcasses from normal animals and packed in bulk.

SALMONELLA BRANDENBURG IN HOME-PRODUCED MEAT AND SAUSAGES

In July 1963, S. brandenburg was isolated from pork sausage meat from a factory (27). A marked increase in the number of isolations was observed in September; 11 to 17 percent of samples of pork and beef sausage meat from two factories, receiving carcasses from the same suppliers, were contaminated with S. brandenburg. Samples of minced beef and pork meat from two other factories showed that 3.6 percent and at least 15 percent, respectively, were contaminated with salmonellae but not S. brandenburg. Sausages of the relevant brand from retail shops were examined in November, and 23 percent of approximately 90 samples were positive. Meat and skin scrapings from pig carcasses from 12 suppliers were examined; carcasses from one supplier only were positive for S. brandenburg on three occasions. In the abattoir concerned, pigs were kept in a number of lairages for periods from 1 day to 1 or even 3 months.

Table 10.—*Types of salmonellae and their frequency of isolation from frozen packed boneless raw horsemeat and horse offal for animal consumption, 1961-63*

Serotype	Country and number of meat and offal samples positive for salmonellae			
	Argentina and Paraguay		Bulgaria, Rumania, Hungary, and Poland	
	Meat	Offal	Meat	Offal
paratyphi B.	7	....	....	....
typhi-murium	15	3	2	....
anatum	316	34	8	4
archaveleta	1	....	....	....
belem	1	1	....	....
bovis morbificans	35	6	1	....
braenderup	.....	1	....	....
brandenburg	1	....	....	....
bredeney	26	1	....	....
chicago	.....	....	....	1
cubana	2	....	....	....
derby	107	9	1	8
dublin	.....	....	1	....
give	86	5	....	....
glostrop	1	....	....	....
good	2	....	....	....
havana	1	....	....	....
infantis	2	1	....	....
kentucky	1	....	....	....
kottbus	.....	....	2	1
meleagridis	410	35	4	2
minnesota	742	99	8	....
mission	1	....	....	....
montevideo	1	....	....	....
muenster	1	....	1	....
newport	43	7	....	....
nottingham	1	....	....	....
oranienburg	321	46	5	....
panama	1	....	....	....
poona	14	5	....	....
saint-paul	1	....	....	....
san-diego	4	....	....	....
senftenberg	15	....	....	....
sundvall	1	....	....	....
worthington	.....	1	1	....
yaba	2	....	2	....
Unnamed	2	1	....	....
Not typed	186	16	....	....
Total samples positive	2,241	265	35	16
Percent samples positive	43.3	51.6	15.3	33.3
Total samples tested	5,179	513	229	48

Table 12 shows that S. brandenburg was isolated from lairages where pigs had been kept before slaughter for 1 to 4 weeks and sometimes longer, in contrast to the predominantly negative results from lairages where pigs were kept for only 2 to 5 days before slaughter.

Table 13 shows the results of examination of mesenteric glands from short and long stay pigs and demonstrates the increase in infection in pigs kept in contaminated lairages for longer than 1 to 2 weeks. It shows also that a very small proportion of pigs coming in from various areas were infected with S. brandenburg indicating a widespread endemicity rather than a focus of infection.

The distributing agent is likely to have been a feeding stuff, but the ill effects may not have been felt in the population had not conditions in one or perhaps more abattoirs encouraged a build-up of infection.

The supply of contaminated carcasses to the two factories involved was stopped and both establishments were cleaned. Isolations of S. brandenburg were considerably reduced but not eliminated; 3.3 percent of 83 samples of pork and beef sausage meat were still positive after these measures had been taken.

Isolations of S. brandenburg from the abattoir lairages were rare after they had been thoroughly cleaned and used for short-stay pigs killed as soon as possible after purchase.

Investigations to find the original source of the infection in the pigs continues. It is hoped that by examining sewer swabs from all the markets from which the carcasses could have come, certain areas and farms where S. brandenburg may be found can be identified. It is likely that there are abattoirs elsewhere where similar foci of infection could develop not only for S. brandenburg, but for other salmonellae also.

COCONUT

Wilson and MacKenzie (28) published a report of an outbreak of typhoid fever caused by S. typhi in desiccated coconut from Papua; incidents of salmonellosis occurred at the same time, and 12 serotypes of salmonellae were found in the coconut. Kovacs (29) isolated salmonellae, including S. paratyphi B, from Ceylonese coconut. From 1959 to 1960, 9 to 10 percent of samples of desiccated coconut examined in the United Kingdom were positive for salmonellae including S. paratyphi B (30). A few cases of paratyphoid fever were reported in children who had eaten marshmallow biscuits coated with desiccated coconut (31), and a mother who ate raw desiccated coconut infected her baby. The paratyphoid bacilli were assumed to have come from human personnel engaged in the manufacturing processes; other salmonellae may have come from human or animal sources. The number of pathogens probably increased during production, but the desiccation process was unable to destroy them all.

Legislation, passed in 1961 by the Ceylon government, led to more hygienic methods of production. Velaudapillai, Nitiananda, and Meedeniva (32) found that 31 of 1,363 (2.3 percent) of samples of desiccated coconut were still positive in 1962, but the United

Table 11.–*Salmonellae in raw meat and offal from butchers' shops, pet shops, knackers' yards (Different areas during 1963)*

Area and Origin	Number of samples examined	Number positive	Percent	Serotype	Information
London, Colindale:					
Butcher shop	91	2	2.2	*typhi-murium* (1), *newport* (1)	Mostly English
Pet shop	61	16	26.2	*typhi-murium* (1), *anatum* (1), *agama* (1), *bovis-morbificans* (1), *enteritidis* (3), *meleagridis* (3), *minnesota* (4), *oranienburg* (2), *senftenberg* (1).	Mostly imported meat
Knacker yard	39	1	2.6	*typhi-murium* (1)	Mostly beef
Bournemouth:					
Butcher shop	2	0	0	...	No information
Pet shop	17	0	0	...	No information
Knacker yard	1	0	0	...	No information
Ipswich:					
Butcher shop	12	0	0	...	Mostly English beef
Pet shop	8	3	37.5	*typhi-murium* (3)	No information
Knacker yard	4	0	0	...	Horse
Leeds:					
Butcher shop	19	0	0	...	No information
Pet shop	20	8	40.0	*typhi-murium* (1), *dublin* (3), *lexington* (4)	No information
Knacker yard	0	0	0	...	
Manchester:					
Butcher shop	50	1	2.0	*typhi-murium* (1), *bredeney* (1)	Mostly English
Pet shop	47	17	36.2	*typhi-murium* (5), *anatum* (2), *bovis-morbificans* (2), *dublin* (3), *enteritidis* (1), *meleagridis* (1).	Mostly butchers' scrap (English)
Knacker yard	3	0	0	*oranienburg* (1), *taskomy* (1), *thompson* (1)	Beef
Newcastle:					
Butcher shop	21	0	0	...	Mostly imported boneless meat
Pet Shop	16	0	0	...	Mostly butchers' scrap
Knacker yard	13	5	38.5	*dublin* (4), *senftenberg* (1)	Beef and horse
Southampton:					
Butcher shop	0	0	0	...	...
Pet shop	45	3	6.7	*give* (2), *thomasville* (1)	Butchers' scrap (mainly English) 32 negative, imported horse 3/13 positive
Knacker yard	2	1	50.0	*bovis-morbificans* (1)	Beef and horse
Total:					
Butcher shop	195	3	1.5		
Pet shop	214	47	21.9		
Knacker yard	62	7	11.2		

Kingdom results for 1963 showed that 1 only of 222 samples (0.4 percent) was positive for salmonellae. Comparative results from desiccated coconut from Ceylon examined in the United Kingdom from 1959 to 1963 are given in Table 14. From 1959 onwards, many U. K. manufacturers of confectionery and bakery products, steam-treated and redried their supplies of desiccated coconut before use (33).

FROZEN SEA FOODS

Approximately 1,650 samples of imported frozen sea foods were examined in the Food Hygiene Laboratory from the beginning of 1962 onwards. The samples were mostly frozen cooked prawns and shrimps, but there were about 200 samples of frozen raw prawns and a few raw lobster tails. Most of the consignments came from Japan, Chile, and China, but other countries were also represented. Salmonellae were isolated from only 4 (0.2 percent) samples. The bacteriological results for these four samples are shown in Table 15. The results indicated that crustaceans were not usually infected with salmonellae, and it was presumed also that the cooked and raw products were rarely contaminated by human carriers.

It is interesting to note that Escherichia coli was not found in 3 of the 4 samples positive for salmonellae; in one sample coli-aerogenes organisms were not found in 0.1 gm.

Table 12.–**Salmonella brandenburg** *and* S. **typhi-murium** *from lairages in relation to length of stay of pigs in abbattoir November-December 1963*

[b = *S. brandenburg;* t-m = *S. typhi-murium;* – = Negative; + = Positive]

Lairage identification number	Occasions of sampling			Market source of pigs	Lairage identification number	Occasions of sampling			Market source of pigs
	1	2	3			1	2	3	
Long stay					Short stay				
4	+ b	+ b	+ b	So	1	–	–	–	M
5	+ b	+ b	+ b	A	2	–	+	–	M
7	–	+ b	+ b, t-m	G	3	–	–	–	A
8	+ b	+ b	Not done	M	6	–	–	–	G
9	–	+ t-m	+ b	Se	19	–	–	–	A
11	–	+ b	–	So	21	–	–	–	Se
14	–	–	–	So	22	–	–	–	A
15	+ t-m	+ t-m	+ b, t-m	So	23	+ b	–	+ t-m	C
16	–	–	Not done	So	24	–	+ b	Not done	Transit
18	+ b	+ b	+ b	C & Se					

Table 13.–*Salmonellae from pig mesenteric glands in relation to contamination of lairage and length of stay of pigs in lairage*

Lairage identification number	Salmonella contamination of lairage	Number of pigs examined	Pigs positive for salmonella		Serotypes isolated from pigs	Market source and length of stay of pigs in lairage
			Number	Percent		
3	negative	19	2	10.5	*brandenburg* *typhi-murium*	A — short
6	negative	19	1	5.3	*brandenburg*	G — short
5	positive: *brandenburg*	19	6	31.6	*brandenourg*	A — long
7	positive: *brandenburg* *typhi-murium*	11	10	90.9	*brandenburg* *typhi-murium* (5 each)	G — long

Table 14.–*Salmonellae from imported desiccated coconut 1959–63*

Year and grade	Number of samples examined	Positive for salmonellae	
		Number	Percent
1959–60: Fine	449	45	10.0
Medium	268	21	7.8
Thread	82	2	2.4
Total	799	68	8.5
1961–62: Fine	643	20	3.1
Medium	586	20	3.4
Thread	27	0	.....
Total	1,256	40	3.2
1963: – Fine	101	1	0.9
Medium	115	0	.....
Thread	6	0	.....
Total	222	1	0.4

Table 15.–*Salmonellae in frozen sea foods, 1962–63*

Sample	Total colony count per gm. at 37° C.	Coliform bacilli present	Serotype
Raw prawns . . . (India)	800,000	0.1 gm. . . . (Non-fecal)	*inpraw.*
Raw prawns . . . (Chinese)	700,000	0.01 gm. . . (Non-fecal)	*derby.*
Cooked shrimps. (Chinese)	45,000,000	0.01 gm. . . (Non-fecal) 0.1 gm. . . . (fecal)	*thompson.*
Cooked prawns . (Chile)	Less than 500	Not found in 0.1 gm.	*typhi-murium.*

CONTROL

A study of salmonellae in food would not be complete without reference to treatment by pasteurization to render the food safe.

The High Temperature Short Time (HTST) method of pasteurization for liquid whole egg has already been mentioned, and it is hoped that a similar method of treatment will be used for liquid egg white. Radiation of frozen whole egg has been described by a number of workers both in the United States (34,35) and in the United Kingdom (36,37). Ley (38) recommended the use of small doses of gamma-rays for the treatment of raw meat, and in particular horsemeat, to kill salmonellae. Thornley (39) and Ley (40) have given technical details and information on the radiation resistance of salmonellae and methods of bacteriological examination after irradiation.

Ley, Freeman, and Hobbs (41) have given the results for the radiation of desiccated coconut and bonemeal as well as of raw meat and liquid egg. The dose required to remove salmonellae from coconut was sufficiently high to cause changes and some rancidity developed. S. typhi-murium was somewhat resistant in bonemeal also. It is not known whether radiation changes in the meal would affect its palatability for animals.

A report on the "Radiation Control of Salmonellae in Food and Feed Products" (42) summarizes the present situation and makes recommendations for the future.

CONCLUSIONS

Egg products, desiccated coconut, and raw meats have proved to be productive sources of salmonellae, and these foods provide the means by which salmonellae can be seeded into the population. The heat-treatment of egg products and more hygienic methods for the manufacture of desiccated coconut have lessened the danger for these two products.

The problem of freeing meat from salmonellae remains. Gamma-irradiation seems to be the most efficient and practicable method of treatment. Animal infection could be reduced by greater care on farms, and in markets and abattoirs. But more attention should be given to animal feeds; contaminated ingredients should be withheld unless methods for killing the salmonellae in the final product can be accepted.

ACKNOWLEDGEMENTS

I wish to thank the Port of London, laboratories of the Public Health Laboratory Service, the Salmonella Reference Laboratory, and in particular, my colleagues in the Food Hygiene Laboratory, for materials, practical work, and collection of data.

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ADDENDUM

THE USE OF PHAGE TYPING IN THE EPIDEMIOLOGY OF SALMONELLOSIS

by Dr. E. S. Anderson[1]

The phage typing of salmonellae has been of immense value in the epidemiological study of food-borne infections due to these organisms. In the case of paratyphoid B fever, the original indications that Chinese egg was the vehicle responsible for carrying the organism came as a result of phage typing. The method has subsequently been used with great effect in the epidemiology of this disease caused by Chinese egg, and its use has contributed to the introduction of regulations requiring pasteurization of liquid egg in the United Kingdom. Similar control was possible through phage typing in the epidemiology of *S. paratyphi* B infection carried by Ceylonese desiccated coconut (1). It was subsequently observed by Anderson (2) that no less than 21 phage types of *S. paratyphi* B were carried by this material. The majority of these types belong to *S. paratyphi* B var. *java* and cause an illness of the food poisoning rather than the enteric fever type.

In the epidemiology of *S. typhi-murium* infections, the results of phage typing have been quite spectacular. Dr. Taylor has described its use in the tracing of infection due to livestock raised in the United Kingdom. But in the tracing of infections due to foodstuffs imported from abroad, it is no less valuable. There are certain phage types of *S. typhi-murium* common in man, but absent, as far as we know, from British livestock. Some of these types have been found in imported foods, and there is a great deal of evidence to support the hypothesis that it is these foods that are responsible for introducing these phage types into England. Anderson (personal communication) has evidence to suggest that the distribution of phage types of *S. typhi-murium* is not the same from country to country; that, for example, types that are rare in England may be common in the United States, and vice versa. Moreover, certain phage types originate almost uniquely from certain countries. When English infections occur due to such types, suspicion is automatically directed to foodstuffs imported from these countries. When *S. typhi-murium* phage typing is used by many countries on a large scale, it will be possible to throw further light on this subject; and it is probable that the epidemiology of *S. typhi-murium* food poisoning will be helped in an international sense.

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Salmonella in Miscellaneous Foods

Mildred M. Galton, Sc.M.[1]

Much of the knowledge concerning sources of human salmonella infection discussed at the Conference thus far has concerned lower animals, their foods, and the basic ingredients of human foods. In recent years, investigations have implicated a wide variety of miscellaneous processed foods as sources of salmonellae (1, 2, 3). Some of these are prepared by adding, to a basic food, an ingredient which may contain salmonellae, such as eggs or meat products; others contain none of the ingredients in which salmonellae are known to be indigenous. One of the common denominators for most of these foods is that they are processed. At a conference in August 1962 (4), on the microbiological quality of foods, a number of papers dealt with problems brought about by changes within the food industry, such as the rapid developments in food technology, including methods of preservation, processing, packaging, and marketing. The failure of microbiological control to keep pace with these rapid changes was recognized, and particular attention was given to research needed in food microbiology to cope with these problems.

Current epidemiological data indicate that food processing plays a major role in the dissemination of salmonellae originating in animals, birds, or human carriers. Several large outbreaks have been traced to processed "ready-to-eat" and to partially cooked poultry meat products. One of the most extensive outbreaks occurred several years ago in Georgia (1) and was due to Salmonella blockley in commercially prepared chicken salad. There were more than 300 confirmed cases and an estimated 3,000 persons developed clinical symptoms. The salad was prepared by one large Atlanta food processor and distributed throughout the State in refrigerated trucks. It was estimated that some 30,000 cartons of contaminated salad were distributed, providing exposure to about 100,000 persons. S. blockley was isolated from recalled samples of the chicken salad prepared on each day of the preceding 30 days. Swab samples taken from materials and equipment in the plant were also positive. Although five workers in the plant were found to be harboring S. blockley and no salmonellae were isolated from the available frozen roosters used in preparing the salad, it was presumed that the organisms were introduced into the plant through infected birds. This conclusion was supported by the occurrence of outbreaks of S. blockley in several broiler flocks in Georgia and South Carolina and by the knowledge that workers in the plant routinely used the plant's prepared products to make sandwiches for their lunch.

After thorough cleaning and sanitizing of the plant under supervision of the Georgia Department of Public Health, adoption of improved sanitary practices and procedures for preparation of salad, and removal of the infected workers from the processing line, the chain of transmission appeared to be broken as no further contamination was found in the product, and no additional cases have been traced to this source.

In 1963, reports were received by the CDC Salmonella Surveillance Unit in which another "further processed" poultry product was implicated as the source of several episodes of salmonella infection. The first of these incidents, investigated by the Pennsylvania State Department of Health, involved four members of one family and the baby sitter (5). All suffered febrile gastroenteritis after eating turkey roll, corned beef, pastrami, and potato salad from a supermarket chain store. S. muenchen was isolated from each of these foods. Subsequently, S. muenchen was isolated from one of three of the individuals handling these foods in the store and from the 13-year-old daughter of one of the food handlers. Both of these persons were asymptomatic. About the same time, three additional cases of S. muenchen infection were found among other customers of the store. Although only seven proved cases of salmonellosis were related to consumption of the contaminated food sold in this supermarket, city health officials estimated that at least 100 undiagnosed cases occurred.

During the S. derby outbreak in hospitals in the northeastern part of the country, two children in Philadelphia, who had no hospital contact, developed gastroenteritis due to S. derby after eating turkey rolls purchased from a local food store. S. derby was recovered from the rolls by the Philadelphia City Health Department laboratory (6).

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An extensive outbreak of food poisoning involving two kitchens at a college in New York State occurred on April 11, 1963, and was reported by the Erie County Health Department and the New York State Department of Public Health (7). Epidemiologic evidence indicated that turkey roll served at dinner on April 10 was the source of infection. It was estimated that 250 students were ill, or 18 percent of those eating in the two dining rooms. *S. manhattan* was isolated from 10 students and 15 of the 31 food handlers. As the two dining halls were in separate buildings and completely independent in regard to equipment, personnel, and facilities, this suggested involvement of a common food served in both dining halls which had been contaminated prior to introduction into the kitchens. All epidemiologic evidence implicated turkey rolls although *S. manhattan* was not isolated from turkey rolls obtained for examination from a local commissary. The rolls were processed by a plant in the Philadelphia area.

Turkey was one of the suspect foods as the possible vehicle of infection in the *S. derby* hospital outbreaks. As turkey rolls had been implicated previously as the source of human infection in three episodes and as they were being served in at least one hospital, materials in the environment of three plants manufacturing this product in one area of Philadelphia were cultured (8). *S. muenchen* was isolated from floor drains in two turkey roll plants and also from the equipment in one of these plants. *S. derby* was found in all three plants and in the finished roll (partially cooked) from one plant. In addition, four other serotypes—*S. saint-paul*, *S. heidelberg*, *S. anatum*, and *S. thompson*—were detected in two of the plants.

After these incidents, the Philadelphia City Health Department issued a regulation prohibiting the sale of partially cooked turkey rolls in the city. According to present U. S. Department of Agriculture requirements, ready-to-eat poultry rolls must be cooked so that they reach an internal temperature of 160° F.

In contrast to prepared poultry products, limited studies of cooked luncheon meats which contain one or more kinds of red meat, have been totally unproductive of salmonellae. No salmonellae were isolated from 287 samples of luncheon meats and cooked casing-covered meats obtained from retail markets in Cincinnati (9, 10), or from 51 miscellaneous luncheon meat samples collected in the Atlanta area (10). Raw products and the environment of two abattoirs in which some of these products were produced were heavily contaminated with salmonellae. The apparent absence of salmonellae in these products may be attributed to several factors. One of these is that they usually contain salts and other preservatives which may discourage growth or even survival of salmonellae after the product has been heated.

Outbreaks of salmonellosis have been traced to sauces and salad dressings contaminated with salmonellae, but these items usually have been found to be "home-made." However, in 1961, a commercially prepared lot of hollandaise sauce was implicated in four incidents of *S. typhi-murium* infection in California and Minnesota. *S. typhi-murium* was isolated from jars of the sauce and from the patients (11).

For many years, bakery products containing eggs, such as eclairs and cream pies, have been implicated as sources of salmonella outbreaks in man (12, 13). This is not surprising when the results of environmental surveys conducted in a single bakery in Wales by Harvey and Phillips (14), are considered. They found salmonellae in 27.9 percent of all samples taken from floor drains in the bakery. In addition, they had no difficulty in isolating salmonellae from the surface of bakery machinery after routine cleaning. In spite of their demonstration of the regular occurrence of contamination in the bakery from 1955 to 1958, they stress the point that there was no major outbreak of salmonellosis infection spread by the bakery products. However, in 1959 Harvey *et al.* (15), reported an extensive outbreak that occurred among school children attending a party. Salmonellae were isolated from 209 of 280 children who had eaten trifles, a cake containing jelly and custard. The same phage types of both *S. typhimurium* and *S. thompson* were isolated from the patients and from the trifles. The only part of the trifle containing egg was the cake. Although this portion was baked at a temperature sufficient to kill the salmonellae, the distribution of these organisms observed in the environment of bakeries in previous studies (14), indicates ample opportunity for contamination after baking.

In the United States, 15 percent of the 96 salmonella outbreaks reported to the Communicable Disease Center during the past 3 years have been associated with custard or cream-filled desserts and bakery products (16). In 1961, stimulated by information received from Dr. Gail M. Dack, Director of the Food Research Institute, University of Chicago, that he had recovered salmonellae from samples of angel food cake-mix purchased on the retail market, we obtained samples of this relatively new type of processed product in addition to several types of bread mixes. Of 22 boxes of these materials from which samples were cultured, salmonellae were isolated from 4 (14%). These products contained dried egg or egg albumen. The types found included *S. tennessee*, *S. infantis*, and *S. oranienburg* (17).

During the investigation of the *S. thompson* outbreak in Michigan in 1962, 42 samples of angel food cake-mix from the Detroit area were examined (18). Salmonellae were isolated from 6 of the 12 packages from one producer. The types detected were *S. tennessee*, *S. infantis*, and *S. anatum*. The two most common types isolated from human food products as reported in the Salmonella Surveillance Reports in 1963 were *S. tennessee* and *S. infantis*. All of the types in these mixes occurred frequently among cultures isolated from man.

Rather extensive studies have been made on all types of prepared cake and bread mixes in Canada (19). Most of these products originated in the United States. Salmonellae were in 55 percent of the samples of angel food mixes in which powdered egg albumen was packaged separately from the rest of the mixture, in 47 percent of the cake mixes containing egg, and in 5 percent of the cake mixes containing no egg. The latter were prepared by the same manufacturer whose angel-food mixes were contaminated. With the exception of *S. thompson*, other types recovered coincided with the types commonly isolated from man and human food products in the United States.

Relatively few human infections have been traced directly to these contaminated cake mixes. In the instances that have occurred, evidence indicated that the cakes were contaminated after baking or the victims "tasted" the uncooked batter, as was the situation with a child in a Newfoundland hospital (20). Skoll and Dillenberg (21) reported an outbreak of *S. thompson* infection which occurred in 7 of 22 men in a highway department road camp. *S. thompson* and *S. heidelberg* were isolated from angel food cake-mix from the camp kitchen and *S. thompson* was recovered from a baked cake prepared from the mix. Since the cake was baked at 375° F. for 30 minutes, it was assumed that contamination occurred after baking from the hands of the cooks, the utensils which had been used in preparing the raw mix, or albumen dust in the environment of the kitchen.

During the past 18 months, a variety of cake and bread mixes have been examined for salmonellae by Adinarayanan (22), and Foltz (23), in Kansas. Salmonellae were recovered from 11 percent of 144 samples of cake mix, bread mix, cookie dough, and dinner rolls. A higher percentage of cookie doughs and dinner rolls were found contaminated than cake mixes or bread mixes. The fact that little difference was observed in the salmonella recovered from products containing egg and from those without eggs, indicated contamination during processing. Again *S. tennessee* and *S. infantis* appeared most commonly. The frequent appearance of *S. tennessee* in processed cake and bread products, suggests that this type may be gaining in prevalence. During the 10-year period 1948 to 1957 in the United States (24), *S. tennessee* ranked 18th among types isolated from domestic fowl; 8th from eggs and egg products; and 7th from human sources. From 1959 in 1961 in England and Wales, it was the 3rd most common type from eggs and egg products (25). Druing a 5-month period in 1962 in Canada, it was the 2nd most common type obtained from egg products and egg-containing foods (26).

Investigation of an extensive epidemic of *S. muenchen* infection among infants in Sweden led to the discovery of these organisms in cereal powder, a type of food product not previously suspected (3). Further, the outbreak illustrated the risks involved with centralized industrial manufacture of foods. Although the chain of proof was not absolute, epidemiologic and bacteriologic evidence warranted the conclusion that a shipment of salmonella-contaminated African barley was the source of the outbreak. During malting, this product infected a lot of Swedish barley. The Swedish product then contaminated the mill used to grind oatmeal, which was used without prior heating in the infants cereal.

The discovery of another previously unsuspected food product, dried inactive yeast, as the source of salmonellosis in hospitalized patients emphasized the importance of considering other processed products when the origin of infection cannot be traced to the usual foods of animal origin or a food handler carrier (2). Three salmonella types—*S. oranienburg*, *S. montevideo*, and *S. senftenberg*—were recovered from five patients suffering from hospital-acquired infections. The same types were isolated from the dried yeast used in the patient's diet. Investigation of the processing plant revealed contamination in the product only during milling and packaging. Source of the contamination was believed to be outside air introduced through a ventilating duct.

It is obvious from the foregoing review that salmonellae may be found in a variety of processed foods regardless of origin. The role of commerical food processing in the distribution of salmonella organisms in meat and egg products is well known. However, the paths of introduction of salmonella into certain other foods are frequently complex. Great changes have occurred in the food industry. Many new products and new methods of processing have been developed. Efforts towards effective prevention of the spread of salmonellosis must be directed not only against control of the disease in the many animal hosts but also in elimination of the sources of contamination in food processing plants. This cannot be accomplished without microbiological testing at each point along the processing line. A monitoring system for salmonellae can best be undertaken by the industries themselves to examine the product before, during, and after processing. It is encouraging to know that progress is being made in this direction. Some industries are establishing microbiological laboratories in their plants; others are conducting active research to investigate problems associated with contamination of their products. Many have expressed their willingness to adopt necessary measures to prevent production of products contaminated with salmonellae as such methods are proved effective.

Training courses are being conducted with the cooperation of government agencies to familiarize food processors with methods for the isolation of salmonellae. In addition, national and international health agencies' programs should include training of those concerned with food microbiology, epidemiological methods for control of salmonellosis, and education of food processors and food handlers in improved sanitary practices.

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Panel Discussion

Dr. Benjamin J. Pomeroy, Chairman,

Mrs. Mildred M. Galton, Dr. William R. Hinshaw,

Dr. Betty C. Hobbs, and Dr. James H. Steele

DR. POMEROY: Before we turn to the panel for discussion, I would like to call upon two men to make a few comments. Doctor H. P. Seeliger of Bonn, Germany, would like to discuss some aspects of salmonellosis as seen in Europe.

DR. SEELIGER: A recent review by Dr. E. H. Kampelmacher (1) deals with the high frequency of salmonella isolations in man in the Netherlands from 1951 to 1960. The total number of serotypes identified rose until 1959; in 1960 there was a slight decrease. The annual number of isolations is now up to 12,000. These figures may also be related to the increased activity of the public health laboratories.

Similar findings have been obtained in West Germany and West Berlin; these data are summarized in Table 1. Salmonella typhi-murium, as in Great Britain, is the major source of salmonellosis, but there has been a decline since 1958. Since that time, the number of other serotypes identified has increased. We have been isolating from man as many as 90 different serotypes in these past years. This is with the exception of S. typhi and S. paratyphi A and B which currently cause about 1,000 to 1,500 cases per year in West Germany.

Salmonellae have been isolated from seafood caught near the mouths of big streams, and there may be a build-up of infectious cycles through seagulls and other birds that take up the infected material from the rivers and streams. Concerning wild life, I believe there are some reports that wild game are occasionally infected by salmonella and maintain their own infectious cycles. There is also a problem with pets, particularly tortoises that are imported from the Balkans, Greece, and Yugoslavia. About 80 percent of them contain salmonella.

DR. POMEROY: I would like to ask Dr. James Williams (Southeast Poultry Research Laboratory, U. S. Department of Agriculture, Athens, Georgia) to make a comment or two concerning the pathogenesis of salmonella infections in poultry.

DR. WILLIAMS: The subject of the epidemiology of salmonellosis is an important one for our consideration. We have reached certain conclusions

Table 1.–*Human salmonella infections in the Federal Republic of Germany and West Berlin, 1953-59*[1]

[Cases of illness and carriers excluding *S. typhi, S paratyphi* A and B]

Year	Total cases	*S. typhi-murium*		*S. enteritidis*		Number of other serotypes
		Number	Percent of total cases	Number	Percent of total cases	
1953	Not known	875		307		38
1954	Not known	975		456		53
1955	Not known	1,674		633		51
1956	6,808	2,610	38	402	5.9	80
1957	5,514	1,919	34	296	5.4	84
1968	4,843	1,988	41	329	6.8	79
1959	5,629	1,941	34	248	4.4	90

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[1] From *The World Problem of Salmonellosis*, Edited by E. L. Van Oye, 1964. Published by Dr. W. Junk, The Hague, Netherlands.

in listening to the data presented so far. One of these conclusions is that poultry, poultry products, and eggs are among the major sources of these infections.

The problem of salmonellosis and some of the difficulties in its control in poultry have been under study for almost 30 years. Here I refer specifically to control on an organized basis through the National Poultry and Turkey Improvement Plans. We now have more than 800 salmonella serotypes, about 120 of which have been identified from poultry. In poultry, this is essentially a disease of young birds less than 1 month old, with the highest mortality occurring from about 7 to 10 days of age.

With reference to the epidemiology of this problem in poultry, the role of the egg in its transmission not only to man, but from bird to bird under natural conditions is of great importance.

It is well established that host-adapted pullorum disease (*Salmonella pullorum*) and fowl typhoid (*S. gallinarum*) are diseases that are usually directly transmitted from the ovarian tissues of infected adults through the egg to the young. Most other salmonella infections are usually transmitted to young birds through penetration of egg shells by organisms on their surfaces, through contaminated feed, or from the many other natural sources of the organisms that abound in environments where chickens and turkeys are raised.

Salmonella organisms may gain entry into the circulatory system and vital organs of the young chick or turkey poult from infection existent within the egg, from consumption of contaminated feed or water after being hatched, or from the inhalation of the organisms in an infected environment. A septicemic state may be established in the young birds, and the salmonella organisms can be readily isolated from such organs as the liver, spleen, or kidneys as well as the intestinal tract. If the impairment of vital organs is severe enough, many of the birds will die. Those that do not succumb may remain fecal carriers of the organisms for long periods of time and may serve to contaminate the surfaces of eggs which they lay. They may also spread the infections to other adults. Adult birds infected with salmonella generally show no outward symptoms.

Buxton (2) called attention to the fact that nutrition may have a significant effect upon the susceptibility of animals to salmonella infections and may be associated with factors concerning the development of immunity in the host and alterations in the virulence of the infecting organism.

The pathological responses of experimentally infected birds show wide variation which is strongly influenced by the serotype of salmonella involved in the infection. Shaffer *et al.* (3), found marked variation in the response of day-old chicks to either peroral or parenteral inoculation with various antigenic types of salmonella. The course of infection produced by *S. typhi-murium* included marked shedding of the organisms in the feces; frequent invasion and localization in tissues such as the spleen, liver, and lungs; and deaths from bacteremia. *Salmonella paratyphi* A exhibited less evidence of invasiveness and caused no mortality. Henderson *et al.* (4), administered seven different salmonella serotypes orally in graduated doses to inbred white Leghorn day-old chicks. All seven serotypes produced mortality varying from about 2 percent (*S. anatum*) to 80 percent (*S. typhi-murium*).

Pathological lesions may be entirely absent in young birds during extremely severe outbreaks. In outbreaks that permit the development of advanced cases, pathological changes include emaciation, dehydration, coagulated yolks, congested liver and spleen with hemorrhagic streaks or pin-point necrotic foci, congested kidneys, and pericarditis with adhesions. Hemorrhagic enteritis involving the duodenum is a common occurrence in turkey poults. Ballantyne (5) reported that in paratyphoid cases which he studied, caseous ceca were found in 33 percent of poults infected with *S. oranienburg*, in 46 percent of poults infected with *S. typhi-murium*, and in 20 percent of poults with *S. newport*. Caseous ceca occurred in 33 percent of chicks with *S. typhi-murium* infection.

Emaciation, necrotic ulcers in the intestines, enlarged liver, spleen or kidneys, nodules on the heart, and distorted ovules may occasionally be noted in salmonella-infected adult chickens and turkeys. Pathological changes in ovarian tissues as a result of paratyphoid infections are not so distinctive or common as observed in the case of chronic carriers of pullorum disease. Chronically infected adults frequently exhibit no lesions. This is particularly true of intestinal carriers. Buxton (6) in discussing host specificity of salmonella strains infecting animals called attention to the need for fundamental information on the chemical pathology of the reactions between the bacteria and host tissues. It was felt that such information would provide a means of explaining the carrier state in salmonella infections.

For pullorum disease and fowl typhoid, we have a very refined serological test through which these infections can be detected in adult carriers. However, since such a test is not yet available for other salmonella infections, we must try to improve the sanitation of the egg itself if the disease is to be eliminated from poultry flocks. For this reason, much of our current thinking on controlling these infections in poultry and, in turn, contributing to their elimination from the human population must involve attention to the egg as soon as possible after it is laid.

A procedure of foremost importance in eliminating salmonella organisms from the shell surface of eggs is preincubation fumigation of the eggs with a high concentration of formaldehyde gas. As soon as possible after being laid, eggs are placed in a tight cabinet and exposed to the fumigant generated by mixing 1.2 ml. of formalin with 0.6 gms. of potassium permanganate for each cubic foot of space in the cabinet. The period of fumigation is 20 minutes. This procedure is being applied on an increasing number of poultry farms in this country at the present time. Results indicate an improved chick quality and other beneficial effect. Its efficacy in eliminating salmonella infections in hatcheries is striking.

Some hatcheries are going to the extent of having all eggs coming into their establishments subjected to high-level preincubation fumigation. At the University of Georgia, in Athens, we have done work in some hatcheries in which *S. montevideo* has become established as an aerosol infection. In some cases, as many as 80 percent of Kauffmann brilliant green plates exposed at various points in the hatchery have been positive for *S. montevideo*. In such hatcheries, fumigation has been utilized very effectively in eliminating the problem. I do not know at this time what role early fumigation may play in the eventual control or elimination of salmonella infections from humans; however, I think that it is something that should be looked into very carefully.

We are also working at Athens on serological tests for the paratyphoid infections as they occur in poultry. Dr. Steele mentioned that these are not effective. However, we are finding that serological tests do have application in the location of infected flocks. We are presently studying a rapid field test for *S. typhi-murium* in which stained antigen is mixed with a drop of fresh chicken blood to detect infected individuals through agglutination. Other procedures being used to detect infected flocks include culturing of the shell surface of hatching eggs and also fecal samples. All of these means are available to us in the field for detecting salmonella infections in an attempt to break the cycle of transmission of this disease. As we proceed with our work on a solution to this problem in poultry, we should thereby be able to contribute to the control of salmonellosis in humans.

DR. POMEROY: We will now begin with the questions.

> **QUESTION:** Comment on the importance of antibiotics in animal feeding stuffs.

DR. STEELE: I do not think that there is any evidence at hand that antibiotics in feed have either controlled salmonella or that they have contributed to the spread of salmonella. There may be some value in the use of the nitrofurans, but there is no value in the use of tetracycline and its derivatives.

DR. POMEROY: In trying to isolate salmonellae from completed feeds, you have to keep in mind these antisalmonella drugs, especially the nitrofurans. In some of the work that was done at Minnesota, a very small quantity of the nitrofuran was found to inhibit the cultures, particularly the broth cultures, and therefore no isolations could be made. However, if high levels of nitrofurans are fed to turkeys and the turkeys are exposed to salmonella, they will still become infected. You may have no mortality, but the birds will be infected, and you will be able to recover salmonella from the intestinal tracts. So once the salmonella and the nitrofurans are together in the intestinal tracts, you have no difficulty isolating the salmonella from the bird; but, in a completed feed, a very small amount of an antisalmonella drug such as 10 grams of furazolidone per ton of feed will prevent the recovery from the completed feed.

> **QUESTION:** Is it not time to recommend that the practice of keeping pet turtles be abandoned? Many States recommend against the sale of Easter chicks and ducklings now as a result of the salmonella problem associated with them. Why not the same with turtles?

DR. STEELE: I do not think that we are in a position to ban an industry until we have studied it thoroughly and can come up with some recommendations as to what the solution may be. I think it is something that can be corrected by the use of feeds that are relatively free of salmonella.

> **QUESTION:** Do you have information on the effectiveness of bacterins in combating salmonella infections in animals, particularly *S. typhi-murium* in calves? Are such bacterin strains specific or might a vaccine prepared against one strain be effective against other strains?

DR. STEELE: I know of nothing that I have encountered in the literature where *S. typhi-murium* bacterins have been used successfully in minimizing disease in calves. In general, I would say there is no evidence to support their value in veterinary practice.

> **QUESTION:** What is the prevalence of salmonella in dairy cattle?

DR. STEELE: The incidence of salmonella in dairy cattle in the United States is relatively low compared to other parts of the world. We do have *S. typhi-murium* that appears in the calves, but by the time the animals reach puberty they throw off these infections. There have been a few investigations in the United States of *S. typhi-murium* in dairy herds on farms where people have become infected. These were thoroughly investigated, and animals were shown to be shedding the organism, without any clinical signs. It did not interfer with lactation, reduce their flesh, or cause abortion in a herd.

In Western Europe, S. dublin is a very serious problem among cattle. In the United States, S. dublin does occur in the far Southwest. If this were an exporting area, we would really have something to be concerned about. If it started moving across this country, we could have a very serious problem in our rural population. I look upon S. dublin as being a serious occupational disease of rural people; it also could break through the barrier and be carried into our meat and meat products. I do not think milk would be an important vehicle because of the way it is handled.

> QUESTION: Do you feel that in recent years there has been an increase in cattle salmonellosis?

DR. EDWARDS: Yes. We see many more cultures from cattle that we did previously, and I think that this increase must be real.

DR. STEELE: There was an interesting report in Veterinary Medicine in February 1964 (7), on an outbreak in North Carolina last September. They describe the outbreak as occurring in a beef herd of 3,000 animals, where some 250 were introduced as feeder stock in September 1963, and trace it through the herd. This is the largest episode of this type that I have heard about in the United States.

> QUESTION: Is it not more correct to say pullorum disease has been eliminated from the breeding flocks than to say that pullorum disease has been eradicated?

DR. HINSHAW: I am sure if Dr. Henry Van Roekel (Department of Veterinary Science, College of Agriculture, University of Massachusetts, Amherst, Massachusetts) were here he would say as far as he is concerned in Massachusetts and in the other New England States where they have tested and found no infection in the breeding flocks, that the commercial flocks are also free of pullorum disease. This follows since the breeding flocks are the source of replacement for the commercial flocks.

DR. POMEROY: This may be true for New England; however, in the Midwest, we have other reservoirs of infection of S. pullorum. Last year, Minnesota instituted regulations so that all show birds would come under the pullorum control program, and out of 253 show birds tested, 20 reactors to the pullorum test were found. These are more reactors than were found in over one million birds tested under the national poultry improvement program. So we do have a reservior of infection that we hope the pullorum eradication program will ultimately eliminate. As the breeding stocks become completely clean, contaminated premises will ultimately become clean, through the die-off of the salmonella organism in the soil.

I could use an illustration that was reported, in cooperation with Dr. L. P. Williams of the Public Health Service, in the Salmonella Surveillance Report Number 19 (8). We investigated one farm that had birds from pullorum-clean sources; and, from among 25 birds tested, we found 11 infected with S. pullorum. So we have a tremendous problem of infection existing and surviving on these farms in the Midwest. It will take years before we have a die-off of these salmonellae even if we do not keep on re-infecting the premises.

> QUESTION: If egg washing increases shell permeability and also spreads contamination from heavily contaminated eggs to all others in the wash bath, should not washing be forbidden?

DR. HINSHAW: There are at least two schools of thought on this egg washing problem. One school feels that we should abolish egg washing and concentrate on getting clean eggs.

As I see it, one could probably say that under some circumstances eggs are washed in an attempt to make grade A eggs out of grade C eggs. The whole purpose is to get the dirt off the eggs and make them acceptable to the public as clean eggs.

Some of the microorganisms do penetrate egg shells; one that has not been mentioned is Pseudomonas aeroginosa. This bacterium is of concern to persons who sell and market eggs as well as to hatchery men. I do not believe that I am in a position to discuss "washing" much more than this. Maybe someone in the audience can add to my brief remarks.

MISS LALLY: There has been a lot of very excellent work done on egg washing which eliminates the submersion of the egg in the wash water. Dr. Wade Brant, Food Technologist, University of California, Davis, California, is now in the process of studying commercial type egg washing procedures. He can be contacted by those who are interested in further information. There are very adequate ways of washing eggs to prevent the spread of contamination from one egg to another and at the same time maintain the quality of the egg.

DR. HINSHAW: I think you have two different problems here. You have the market egg situation and you have the problem of eggs being produced for breeding purposes. Those who are using eggs for breeding purposes are more prone to say that "we do not want washed eggs" than those who are using them for market purposes.

Many in this audience use eggs for propogation of viruses, and this group certainly is against egg washing.

DR. CARPENTER: Field tests are currently being done that show that we may expect from 1.8 to 2.0 percent better hatchability if all eggs are washed. These observations were made over a period of months in Arkansas and will be reported in the literature.

FROM THE FLOOR: I would like to add one point concerning adequate procedures for washing. This matter cannot be resolved entirely on the point of contamination with pseudomonas or salmonella organisms. If there are adequate procedures, you will also have to take into consideration the economics of the entire picture.

DR. FORSYTHE: I would like to make a comment on this. One must consider the surface contamination of what appears to be a clean object. I am sure all of you are familiar with this. You will find considerable numbers of bacteria on the surface of clean grade A eggs. This has been well established in the literature, and I am surprised that you would even consider not washing eggs as a step in the right direction. We have to wash them properly just like you have to have proper sanitation procedures in hospitals. I am sure you would not consider stopping the surgeon washing his hands because he might do a bad job occasionally. We must improve the methods used.

> QUESTION: How do you explain the indifference to animal manure on salad crops since it was recognized early that human manure was an important public health problem when used to fertilize fresh vegetables used as salad ingredients?

DR. HOBBS: In the first place the danger from salad crops is not disregarded. The washing of salad vegetables in a solution of sodium hypochlorite (60-80 p.p.m.) has been recommended for some time. Results from work carried out on a cruise ship indicated that there were large numbers of Escherichia coli on the lettuces shipped from various ports; the wash waters were not examined for salmonellae. Using this concentration of chlorine (60-80 p.p.m.) in the wash water reduced the E. coli count to a minimum.

The second point is the relative significance of animal and human manure. I assume that human manure is far more dangerous because it may contain typhoid bacilli which are infective in very small doses. Whereas, animal manure is unlikely to contain S. typhi and probably S. paratyphi B, it may contain other salmonella serotypes, most of which are infective in large doses only. Thus, the lettuce leaf carrying small numbers of salmonellae other than typhoid bacilli, is unlikely to cause infection unless it is used for garnishing, when the organisms can multiply in other ingredients of the salad.

> QUESTION: What regulations might an egg processor be asked to follow in the elimination and control of salmonellae from his product?

DR. HOBBS: I assume the query is related to hen egg products only, but it also would be interesting to know the incidence of salmonella in turkey eggs, for example. We know that ducks' eggs have a high rate of contamination, but I presume that, officially, it is not permitted to include ducks' eggs in breaking plants. The proportion of contaminated samples taken in egg processing plants must vary from time to time. Investigating a United Kingdom breaking plant not long ago, we found that for 2 to 3 weeks samples might be free from salmonellae and then batches of samples might be contaminated to the extent of 30 percent, usually with either S. thompson or S. typhi-murium. Thus, there were alternate periods of clean and contaminated eggs. It was obvious that the source of the salmonellae was the eggs and that the farm, the birds, and probably their feeding stuffs were all concerned. Thus, it would be impossible to pronounce any processing plant free from salmonellae unless samples had been examined over a long period of time. However good the hygiene of the plant may be, it can never be free from salmonellae coming in via the eggs themselves. The results of examination of sewer swabs from broiler plants support this opinion. There may be months of freedom from salmonellae followed by a run of S. thompson, S. typhi-murium, and other serotypes.

The use of cracked eggs increases the hazard from salmonellae. I do not know whether the washing or fumigation of eggs may reduce contamination, but it has been said that washed eggs are more permeable to microorganisms. I do not believe that any measures of hygiene, except pasteurization, within the processing plant could be wholly effective against salmonellae. As of January 1, 1964, a statutory regulation in the United Kingdom requires that liquid whole egg for human consumption must be pasteurized at 148°F. (64°C.) for 2-1/2 minutes and that the pasteurized product should be checked for efficiency of treatment by the alpha-amylase test.

MR. HENNINGSEN: You mentioned the alpha-amylase test previously, and gave two examples (page 85). There was one from Australia, where they did the pasteurization and the product passed the alpha-amylase test, but it was positive for salmonella. Secondly, the Chinese products failed the alpha-amylase test but were negative for salmonellae; how do we correlate these examples?

DR. HOBBS: It is probable that the Australian egg was contaminated after pastuerization. Later samples received on a number of occasions from the same source were satisfactory. For the next year or two, we will continue to examine samples of pasteurized liquid egg for salmonellae; and in addition will carry out the alpha-amylase test, although the salmonella investigation is not required by the regulations. It is probable that the Chinese are underprocessing by heating at a lower temperature for long enough to destroy the salmonellae but not to inactivate the alpha-amylase. Whole egg powder could be rendered safe by pasteurizing the liquid egg mix before drying, but unfortunately the alpha-amylase test cannot be applied to the spray-dryed product because the enzyme is affected by the heat of spray-drying. Thus, pasteurization regulations for spray-dried whole egg cannot be instituted until a test other than bacteriological examination is available.

There is a similar problem concerning egg white which does not contain alpha-amylase. Investigators in Cambridge are searching for another enzyme or similar substance which will be inactivated at the temperature found to be convenient for pasteurization. The Danes are already exporting pasteurized frozen liquid egg white which appears to be satisfactory for usage at bakers or confectioners. In the United Kingdom, we are starting experiments on the heat-treatment of egg white.

QUESTION: The practical experience data showed some failures in the 148°F. (65°C.) pasteurization treatment of the egg products in the United Kingdom. This was, I believe, assumed to be due to faulty processing at this beginning stage. Are there any commercial results showing that treatment at 148°F. for 2-1/2 minutes gives less failure than 143°F. (62°C.) for 3-1/2 minutes or some similarly milder and possibly easier to perfect treatment?

DR. HOBBS: The reason that we found salmonellae in one batch of the Australian pasteurized egg, said to be heat-treated at 148°F. (65°C.) for 2-1/2 minutes, must have been either post-processing contamination or failure to realize that in the early stages of a pasteurization run the temperature may not be correctly adjusted, and that this material should be reprocessed.

We decided to recommend a temperature of 148°F. for 2-1/2 minutes because it was generally agreed that the shorter the time of heating, the better the physical properties of the product. Various time-temperature combinations were tried in a commercial plant, but we realized that there might be slight variations in the recorded temperatures. Therefore we carried out laboratory experiments with small quantities of egg in metal "U" tubes fitted with thermocouples. This gave a precise measurement of temperature. Using this technique, we found that 148°F. for 2-1/2 minutes destroyed S. senftenberg, although lower temperatures were adequate for S. typhi-murium. We decided that for safety purposes it would be advisable to use the higher temperature providing the product was acceptable to the baker. It was a coincidental finding that alpha-amylase was also destroyed at a temperature of 148°F. applied for 2-1/2 minutes. It may have been thought that a temperature of 148°F. was recommended to fit the alpha-amylase test, but this is not true. Salmonella typhi-murium was destroyed at 143°F. (62°C.); and, if held for a long enough time at this temperature, S. senftenberg might even be destroyed. However, long heating periods are not good for the physiological properties of the egg; furthermore, our Ministry of Health requires that we have a biochemical rather than a bacteriological test to prove the efficiency of pasteurization.

In practice, both pasteurization and the alpha-amylase test are working well in the United Kingdom, where there are approximately 30 egg processing plants in operation. Pasteurizing under the same conditions is being done in Australia, China, South Africa, Israel, and on the Continent.

QUESTION: Would you recommend a regulation requiring a food handler (e.g. a worker in an egg processing plant) to be examined for salmonellae; and, if so, how often should this examination be performed?

DR. HOBBS: This is surely unnecessary in view of our knowledge of the incidence of salmonellae in the eggs. Workers may become infected while working with egg products, but their possible contribution of salmonellae to the mix will be negligible compared with the eggs as a source. In the United Kingdom, we recommend that food handlers working on hazardous procedures involving, for example, the preparation of cream cakes and the insertion of gelatin into pies, should be checked for salmonella at first employment and perhaps at regular periods thereafter; although there is general agreement that there should be pre-employment examination, re-examination at regular intervals has been a matter for discussion.

QUESTION: Will meringue containing egg whites support the rapid growth of salmonellae? If so, under what conditions?

DR. HOBBS: It is well known that salmonellae multiply in egg white, and they have grown in pan-dried albumen slowly drying at 104°F. (40°C.).

The average concentration of sugar in meringue is about 10 percent, and such a mixture would encourage the growth of salmonellae under proper temperature conditions and at a pH above approximately 4.5.

> **QUESTION:** Does hard freezing affect the salmonella in beef?

DR. HOBBS: Although the samples of boneless beef and other meats which we receive have come from hard frozen blocks, we have been able to recover salmonellae from them. The very act of freezing will reduce the count and kill some of the salmonellae, but there are many which survive. We have stored frozen whole egg contaminated with *S. paratyphi* B for years, and we have still been able to isolate paratyphoid bacilli. The numbers may drop steadily, but it would be almost impossible to say when the last bacillus has died.

> **QUESTION:** If the high proportion of positive samples of meat imported into the United Kingdom is due to poor abattoir hygiene, how do you explain the wide difference between veal and beef imported from New Zealand?

DR. HOBBS: This is an interesting point. Frozen boneless veal from New Zealand has been more heavily contaminated, particularly with *S. typhi-murium*, than the frozen boneless beef, and the reverse is true for Australia. One explanation might be concerned with the relative quantities produced. New Zealand exports a lot of veal and little beef, while Australia exports much beef and little veal. In New Zealand large numbers of calves are brought together for slaughter. This procedure probably involves long distances for transport and long periods when calves are herded together. The small number of calves slaughtered in Australia would not entail so much transmission of infection from one calf to another.

Dr. Peter Nottingham from the New Zealand Meat Research Institute has suggested that there was in fact a variation between meat packing stations in New Zealand according to the number of animals handled. In some of the smaller stations, the animals are transported directly from the farm to the packing station. The meat from these stations was low in salmonella contamination; but, in the bigger stations handling a large number of animals which had traveled a long distance, the salmonella problem was much greater. Presumably there would be a similar situation between large and small stations with regard to beef cattle.

FROM THE FLOOR: New Zealand veal is primarily from bull calves killed only a few days after birth; they are not fed and this may increase their susceptibility to infection.

> **QUESTION:** (1) What temperature should food be kept between serving? (2) What is the safe time allowed between thawing of food and cooking?

DR. HOBBS: It is recommended that food which must be stored hot should be kept at a temperature not lower than 145°F. (63°C.). This is particularly relevant for the storage of cooked meats to prevent the germination of spores of Clostridium perfringens. Food Hygiene Regulations in the United Kingdom require that all cooked foodstuffs served to the public should be kept, between cooking and consumption, either cold (domestic refrigeration) below 50°F. (10°C.) or hot, above 145°F.(63°C.). After cooking and before refrigeration, the time of storage at atmospheric temperature should not be longer than 1-1/2 hours. The same principles can be applied to the time between the thawing of food and cooking, although this period is not so hazardous in view of the subsequent heat-treatment.

> **QUESTION:** Are salmonellae found in the milk of animals and has there ever been an outbreak of salmonellosis from milk?

MRS. GALTON: Salmonellae have been found in the milk of animals, and outbreaks have been attributed to milk. However, for the most part, presence of salmonellae in the milk has been traced to fecal contamination, or to contamination by human carriers. There have been a few rather large outbreaks attributed to milk contaminated with *S. typhi-murium*, with *S. dublin*, as well as with other serotypes.

DR. LEVINE: Are we to infer that the number of cultures found in the Communicable Disease Center laboratory examination is a measure of the relative incidence of the infections in our food producing animals?

MRS. GALTON: I am not sure just what cultures are being referred to. We do isolate and examine some cultures in our laboratory, but they usually concern specific investigations. Most of the cultures that are received now in the Enteric Laboratory, CDC, are from human sources. Those from non-human sources are typed at the National Animal Disease Laboratory at Ames, Iowa. If we look at the types reported from animals both at the National Animal Disease Laboratory and those isolated in our laboratory, it may produce bias because of concentrated investigations in specific areas stimulated by specific incidents. We really have no complete data on the true incidence or prevalence of salmonellae in our animal population.

DR. LEVINE: I would like to point out that in most reports of nonhuman isolations, the majority of isolates are from poultry. This might give people a wrong impression as to the relative incidence of salmonellosis in poultry as compared to salmonella incidence in other animals. I think that the bird, undeservedly, is made the scapegoat for a large part of the salmonella problem.

Probably more fowl are examined bacteriologically in laboratories than all other animals put together, including humans. The basic training of a poultry pathologist I think still is, to a certain extent, that of a bacteriologist. Poultry biologists have been accused of having as their sole armamentarium an inoculating loop; whereas I accuse the veterinary pathologist of having as his sole instrument, the microtome. It is not easy to take cultures from a large animal that is sick or dying in the field; however, most poultry are taken to the laboratories. We have a tendency to interpret these figures incorrectly and equate number of cultures submitted to the laboratory with actual incidence in the field.

The risk of human transmission might be measured by the weight of the infected food animal. Salmonellae have been found in beef, pork, veal, and in chickens. Let us compare the 5-pound chicken with the 1200-pound steer. It might not be too unrealistic to consider the risk of salmonella infection from a beef as 200 times that from a chicken.

MRS. GALTON: Dr. Steele mentioned this morning that because more poultry were examined this may well influence the high prevalence of salmonellosis that we see in poultry.

DR. BRANDLY: I feel called upon to defend the meat industry. I know you are loyal to the poultry industry, and I have worked with poultry, too. Certainly, there is much to be said in what you have observed.

I can only relate my own experiences in the detection of salmonellae in the animal. I think there is a lot to be considered when you start to think about what the housewife does with the meat and the possible spread of the organism to the family if there are salmonellae on it. I want to congratulate you on your discretion, too, in recognizing that poultry pathologists are primarily bacteriologists and that the large animal diagnosticians resort to just pathology, inoculations, serological tests, and so on. When I handle chickens, my hands are not greasy and it is easy to rinse them, maybe wipe them off and that is the end of it. If you are handling red meats — fat steaks, hamburger, pork, sausage, and that sort of thing — you scrub your hands well with soap and wash them off before going on with the next thing; and certainly the contamination of other foods from red meats is just naturally going to be a lot less than it is if you have a product like chickens. The other thing that I have failed to recognize in my pursuit of the culinary art is the recommendation that we eat chicken raw. The gourmet usually recommends that we just sear beef and eat.

If we had a significant salmonellae level in the red meats that we eat in the United States, no one would be recognizing or recommending this practice.

> QUESTION: Comment on the relative hazards involved in the use of stuffed versus non-stuffed frozen turkeys with reference to the temperature lag in thawing and cooking.

DR. ANGELOTTI: We have no first hand information on the subject as a result of work performed in our own laboratories. However, the data collected by Drs. Rogers and Gunderson in 1958 (9) on the survival of salmonellae and staphylococci during the roasting of frozen stuffed turkeys revealed that the lag time required for the slowest heating portion of a 14-pound, stuffed, frozen turkey to reach 165° F. (74° C.) was 7-1/2 hours. By means of inoculation studies in stuffed birds, it has been shown that an internal temperature of 165° F. (74° C.) had to be reached before salmonellae were no longer recovered.

MRS. GALTON: I might add that there have been many reports of episodes of food infection traced to roast turkey.

> QUESTION: What evidence was there from the investigations of the *S. blockley* outbreak in Georgia to indicate the source of the contamination?

DR. McCROAN: The presenting fact in the *S. blockley* investigation was no more than an undue incidence of this serotype reported by the laboratory. When we were following this lead interviewing one of the persons from whom *S. blockley* was recovered, one gave a history of eating commercial chicken salad, a food unusual for her, prior to the onset of her illness. With this information, it was easy to return to the patients already questioned and verify the fact that they also had eaten chicken salad prepared by the same large Atlanta processor, who distributed the product by refrigerated trucks to retail stores throughout Georgia.

An unusual part of the retrospective investigation was the availability of the salad itself. Despite a projected shelf-life of 7 days, specimens withdrawn at our request from display for sale in markets were as much as 30 days old. *Salmonella blockley* was isolated from each sample tested. Observation indicated that there had been no increase in numbers of salmonellae with age; and, although we did not quantitate it, the highest numbers of organisms were found in fresh specimens.

The processing plant was then visited and four assembly line workers were found to be harboring *S. blockley*. Environmental cultures from the plant were also positive. It was observed that some of the same employees who cut up the raw birds moved down the assembly line, still wearing the same torn gloves and bloody clothes, where they boned the cooked chickens and recontaminated the meat. Removal of the four carriers and a thorough cleaning of the environment and a change in the movement of employees was accomplished. Several new batches of chicken salad were prepared and again were shown to be contaminated with *S. blockley*. At this time, another carrier was located and removed. After this, no further contamination of the chicken salad was demonstrated.

Salmonella blockley was not isolated from the frozen broilers sampled in the plant; however, several months later a sizable *S. blockley* outbreak caused unusual mortality among several broiler flocks in Georgia and South Carolina. Thus, there is strong presumptive evidence that the organism was introduced into the plant through infected birds; the employees became infected by handling the birds during the production process and also by eating the chicken salad at noon. The cooked final product was then recontaminated by the workers.

> **QUESTION:** To what degree and to what extent in a case of food poisoning are examinations made for the presence of organisms other than salmonella?

DR. McCROAN: The first thing we do is try to define time intervals well enough to compute a median incubation period, and to determine from this and the symptoms reported by a representative group of patients, the probable etiology of the epidemic. Any well-run diagnostic laboratory will demand this information, and food specimens will then be examined in most larger laboratories for any organism which might conceivably be the etiologic agent. A satisfactory finding is achieved only when epidemiologic, clinical, and laboratory findings agree. Parenthetically, it is not unusual to find a salmonella which is only an innocent bystander and is not the cause of the outbreak.

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INTERSTATE OUTBREAK OF *SALMONELLA DERBY* GASTROENTERITIS

MODERATOR : ***Dr. Alexander D. Langmuir***

Epidemiological Investigation of the Outbreak

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Introduction. An epidemic of infections due to Salmonella derby began in March 1963 and has continued to the present (March 1964). It has involved patients or employees of hospitals or chronic-care institutions located primarily in the northeastern United States. S. derby infections are believed to have been acquired initially through a vehicle common to each of the involved institutions. Once introduced, the infecting organism frequently persisted in both the hospital population and environment. Each of these may have then served as foci for secondary spread of infection. The true extent of this epidemic cannot yet be determined, but it has clearly become the largest outbreak of hospital-associated salmonella infections recorded to date.

The purpose of this presentation is fourfold: (1) to record the chronology of the outbreak, (2) to identify those circumstances and events that may have contributed to the genesis and perpetuation of this outbreak, (3) to describe the current status of the outbreak, and (4) to offer inferences concerning the nature of interrelationships of host, parasite, and environment that may broaden or alter our concepts of the epidemiology of salmonellosis.

Method of Study. Investigation of the outbreak from its inception to the present has primarily been a cooperative effort among hospital staffs and personnel and various health, agriculture, and food and drug agencies at all levels of government. Segments of industry cooperated by permitting appropriate field studies and, in certain specific instances, actively participated in the studies. The Communicable Disease Center (CDC) functioned as central clearinghouse for data necessary to a coordinated epidemiological investigation. During the first several months of the outbreak, investigators in certain locales compiled daily progress reports. Nationally, summaries of the investigation, including tabulations of reported human and nonhuman isolations of S. derby were prepared monthly. "Cases" were tabulated nationally on the basis of bacteriological recoveries of S. derby, without regard to the presence or absence of symptoms. Thus, unless otherwise specified, "case," "infection," or "isolation" are synonymous. The investigation may be considered as having proceeded in two phases which, though continuous and often overlapping, are identified for convenience of presentation: (1) study of initially occurring cases, considered primary infections and (2) identification and study of presumed secondary cases. The rationale for this division will become apparent subsequently.

The Outbreak. Isolation of S. derby from five patients in a single New York City Hospital between March 16 and 29, 1963, was the first indication of an outbreak. S. derby had previously accounted for only 2.0 percent of total salmonellae isolated annually, which is equivalent to approximately 20 isolations per month from the entire United States. Study of the New York City cases was begun in early April 1963 by the New York City Health Department and hospital authorities with CDC assistance. All five patients from whom S. derby was isolated had been recovering from extensive abdominal surgery when severe salmonella gastroenteritis developed. Illness occurred 7-10 days postoperatively, when each patient had been receiving only a highly restricted feeding consisting of commercial preparations of fat and protein and eggnog. Since these patients had been exposed to no other single possible source of salmonellae in common, the investigators concluded that one of the ingredients in the feeding had been the vehicle of infection in this apparently limited outbreak. On April 25, the Philadelphia Department of Public Health reported to the CDC that approximately 20 cases of S. derby gastroenteritis had occurred in a single metropolitan hospital during the preceding 4 to 6 weeks. By May 11, four additional hospitals were reported to have a similar problem. At this point, intensive epidemiological field investigation of each S. derby isolation was begun, and daily surveillance of newly reported infection was undertaken.

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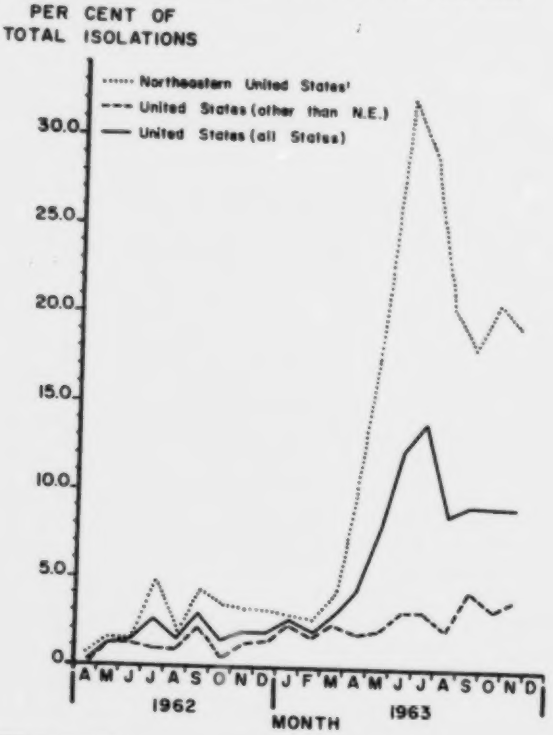
Figure 1.–*Percent* Salmonella derby *isolations of total salmonellae isolations, March 1–Nov. 29, 1963*



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[1] Includes Maine, New Hampshire, Vermont, Massachusetts, Connecticut, Rhode Island, New York, Pennsylvania, New Jersey, Maryland, and Delaware.

Each State health department was requested to investigate and report all isolations of S. derby to the CDC. Epidemiological and laboratory field assistance was made available to the States.

While the investigation proceeded, the number of isolations of S. derby continued to mount. By mid-June 1963, nearly 400 hospital-acquired infections had been identified. The epidemic curve (Figure 1) shows reported S. derby isolations by month. Isolations are expressed as percentages of total salmonella isolations reported to the CDC by the northeastern States and the 50 States, respectively. The initial peak of the epidemic appears to have occurred sometime during June and July. This is undoubtedly subject to some bias because surveillance was most intense, and several mass culture surveys were performed during this period. In addition, there was a lag in reporting in some areas of perhaps 2 to 3 weeks. By mid-July, the most likely common source of infection had been identified, and presumed secondary spread of infection had become a major problem. In August, an abrupt decline in reported cases occurred; a plateau of approximately 175 new cases per month was reached and maintained until December 1963 when a second peak was noted in the epidemic curve. This latter increase in cases will be considered as a separate entity later.

In summary, as of now, (March 1964) the epidemic has spanned an 11-month period during which most prominent features of the epidemic curve have been: (1) an abrupt onset with a rapid rise to a peak incidence of cases, (2) an almost equally rapid decline to a plateau, (3) maintenance of the plateau, far above normal incidence of S. derby isolations for 3 to 4 months, and (4) an abrupt second peak of reported isolations. During this entire period the number of isolations of S. derby reported as not hospital-associated has remained within the range expected during a non-epidemic period, approximately 20 per month (vide supra).

The Investigation: Epidemiology of Initial Cases and Implication of a Common Source of Infection. Clinical histories and epidemiologic data were obtained from 369 of the 372 hospital-associated cases, representing 22 hospitals, that were reported between March 1 and June 21, 1963. Some of these cases were thought possibly to have resulted from person-to-person spread of infection and were thus considered secondary. In order to study a group of purely primary cases, only those with S. derby isolated prior to May 1 were chosen for presentation here. This provides a group of 55 cases which represent 12 hospitals located in four States: New York, Pennsylvania, New Jersey, and Indiana. Each patient had experienced gastrointestinal symptoms compatible with salmonellosis. Fifty were hospitalized patients and five were hospital employees. Their ages ranged from 9 months to 86 years with a median of 49 years and a modal age group of 60-69 years. Sex distribution favored females in a ratio of 2:1. Patients had been hospitalized 2 days to 2 years prior to onset of symptoms; 38 percent of onsets occurred in the first week of hospitalization; 36 percent in the second week; and 26 percent after the second week. Dates of onset of illness occurred randomly from March 3 to April 30. Onset of illness occurred postoperatively in 22 patients, and preoperatively in 6; 27 of the group (including the employees) had not had surgery. A striking incidence of antecedent gastrointestinal disease and maliganancy was noted. Thirty (54.5 percent) cases had gastrointestinal disease, and 11 (20 percent) had a diagnosed malignant neoplasm as major cause of their hospitalization. Past experience has indicated that patients with these diseases have an increased susceptibility to salmonellosis. Of 23 patients in the study group for whom X-ray data were available, only 6 had gastrointestinal diagnostic radiology prior to onset of illness. Following careful investigation, a possible role in dissemination of the S. derby organism could not be assigned to the employees included in the study group.

As the 55 selected primary cases were investigated, a recognizable pattern to their occurrence emerged. Individual cases, though found primarily among a highly susceptible population, occurred sporadically in both time and location within the hospital. This pattern of appearance of cases permitted certain conclusions about the source of the epidemic. It strongly suggested a common source of infection, but clearly indicated that primary infection had not been acquired by contact with possibly infected persons or common exposure in specific locations.

The appearance of these cases in many hospitals in several States incriminated a common source of infection involved in interstate movement. The following possibilities were considered: (1) interhospital exchange of patients or personnel, (2) use of diagnostic or therapeutic agents, and (3) dietary items.

Intensive investigations of each of these possibilities were conducted among the first 369 cases reported from 22 hospitals. Personnel and admission records were examined in the six hospitals that first reported infections. No interchange of patients or employees had occurred during the first month of the epidemic or in the two preceding months. Thus, the first possible common source was eliminated. Attention was then directed to the use of diagnostic or therapeutic agents. Since many of the patients had undergone surgery or X-ray examination, circumstances surrounding the use of each of these techniques were defined for all patients studied. As has been indicated, no procedure or associated diagnostic or therapeutic device was common to a sufficient number of patients to warrant further investigation. Medications such as analgesics, cathartics, and sedatives were administered to a large number of these patients. However, once generically identified, each could clearly be excluded. Thus, the second group of possible sources could be eliminated, and investigation of dietary items was undertaken. This category of sources presented a wide range of potentially suspect foods. Histories of foods or dietary supplements consumed during the 48 hours prior to onset by patients with primary cases revealed that most were receiving highly restricted diets. Thus, many food items could be excluded from further consideration. Only one particular food item of those remaining was almost universally consumed by the patients. This was raw or undercooked eggs. Since eggs are often included in a variety of hospital diets, studies were conducted in three hospitals to determine the normal frequency of their use for comparison. These investigations revealed that approximately 50 percent of the total population could receive raw or undercooked eggs in any given 48-hour period. Among the 55 primary cases presented, diet histories were available for 51; 49 (96 percent) of these had consumed raw or undercooked eggs. A similarly high percentage (89 percent) was noted among the first 369 reported cases. Therefore, a field investigation of egg sources was undertaken.

The field investigation is the subject of a separate communication, and will be only briefly considered here. A careful search was made for the sources of eggs supplied to 15 of the involved hospitals in three States. This involved tracing eggs through large distributors, such as dairies; to a variety of wholesalers; to regional processors; and eventually to individual farms. It was learned that perhaps 12,000 poultry farms could have supplied one or more of the involved hospitals. Among this number of farms only a limited number in two localized geographic areas could possibly have supplied all 15 hospitals.

Samples of eggs (235 dozen), pooled chicken droppings (5,400 specimens), and poultry feed (58 specimens) were obtained in both areas by State, local, Food and Drug Administration, and CDC personnel. These samples were cultured in the Veterinary Public Health Laboratory at CDC. *S. derby* was recovered from 4 of 42 slurries (15 to 18 whole eggs per slurry) of cracked or "checked" eggs and one sample of feed. No salmonellae were recovered from grades A or B intact eggs. Attempts to trace the organism further have been completed and will be reported subsequently.

Recovery of *S. derby* from 10 percent of cracked egg slurries is highly significant. In a culture survey of cracked eggs taken at random, one would not expect to find this degree of salmonella contamination (1). Further, if such contamination were found by chance, recovery of a variety of salmonella serotypes other than *S. derby* would be expected (1,2,3,4). Failure to recover *S. derby* from grades A and B eggs is not surprising. Though such eggs may become contaminated, they do so less frequently than cracked eggs (5). In addition, the bacteriological examination of large numbers of eggs did not begin until the third month of the outbreak, thus possibly decreasing chances of recovering an organism that may have been prevalent earlier.

The evidence presented clearly indicates a source of infection common to each of the hospitals initially involved. Epidemiological evidence with convincing laboratory support strongly suggests that raw or undercooked eggs were the source of primary infections in this outbreak.

Early in the course of the above investigations, two "community-acquired" cases of *S. derby* gastroenteritis were reported from Philadelphia. Both cases were traced to consumption of a contaminated turkey roll. Environmental examination of three turkey roll processing plants in the Philadelphia area revealed the presence of *S. derby* in each. It is significant that, in one of the plants, *S. derby* was the only salmonella organism recovered. After this lead, a bacteriological surveillance of turkeys supplied to a large Philadelphia hospital involved in the outbreak was undertaken. Over 150 turkeys have been cultured by the hospital laboratory with CDC confirmation. Eleven salmonella organisms have been recovered, none of which is *S. derby*. As yet, there is no convincing evidence to link turkey or turkey rolls to the hospital-associated epidemic.

Clinical Studies and Secondary Spread of Infection. Successful clinical evaluation of infections requires the study of large numbers of cases. To meet this need, a coordinated clinical and epidemiological study of *S. derby* infections was conducted in a large general hospital where over 500 infections occurred. Results of this study will be presented in a separate report. It should be emphasized that although infections appeared initially according to a highly repetitious pattern in nearly all involved hospitals, the subsequent course of events was by no means similar in each hospital. Therefore, generalization concerning events subsequent to introduction of the organism is difficult. However, certain distinctive features existed, and these will be emphasized.

On July 11, 1963, the Surgeon General of the U. S. Public Health Service recommended that use of raw or undercooked eggs be discontinued in hospitals. All hospitals involved in the *S. derby* outbreak complied. At approximately this time, nearly two-thirds of involved hospitals ceased reporting new isolations of *S. derby*. In at least two of these hospitals, the disappearance of *S. derby* infections has been confirmed by bacteriological survey of segments of the patient and employee population. However, the remaining one-third of hospitals continued to experience new cases. These hospitals had, in general, been involved earliest in the outbreak and had experienced a larger number of cases prior to the peak of the epidemic. In retrospect, data suggest that the existence of six or more simultaneous *S. derby* infections in an institution predisposed to continued appearance of new cases for the duration of the epidemic. These observations indicated that a critical level of *S. derby* infections might have existed, beyond which persistence of the organism and secondary spread of infection were inevitable.

Certain studies proceeding concurrently, confirmed that this suggestion was, in fact, correct and that a sufficiently large number of patients did serve as a focus for environmental contamination and secondary spread of infection. That environmental contamination occurred is a matter of record (6). In several hospitals with a persistent problem *S. derby* was recovered from a variety of fomites and hospital air. Certain geographic locations within hospitals appeared as repeated foci for new cases, and the temporal occurrence of new symptomatic cases generally became less random. The apparent predilection of *S. derby* for patients with gastrointestinal disease became less marked (54.5 percent among primary cases and 26.9 percent among 272 presumed secondary cases in one hospital). Extensive bacteriologic surveys by rectal swab technique were conducted among patients and employees in several hospitals. Conclusions reached from results of employee cultures strongly support the concept of secondary spread of infection. In general, in only those hospitals experiencing persistence of *S. derby* infection were a significant number of employees shown to be infected. Further, in institutions where large numbers of employees were infected, serial cultures indicated that those employees (student nurses and practical nurses) whose patient contact was most frequent and intimate were infected earliest. However, after a period of several months, infection rates among employee groups were approximately equal.

Thus, the weight of epidemiological and laboratory evidence indicates that secondary spread of infection occurred. The precise mechanisms by which this spread occurred have been difficult to determine conclusively. Most likely possibilities are the following: (1) person-to-person spread by contact, or (2) person-to-person spread via fomites or aerosol. Examples of cases in which each of these mechanisms has been thought operative will be included in a separate report. The omission of food as a possible vehicle of secondary spread is based upon convincing epidemiological and laboratory evidence. The epidemiological pattern (vide supra) was that of person-to-person spread rather than that of a common source outbreak. Investigation of the small clusters of cases, apparently related geographically and temporally, consistently failed to show an association with type of diet or dietary items. Further, stool culture surveys conducted among hospital employees have repeatedly shown foodhandlers to be among employee groups infected latest in the outbreak and with lowest over-all infection rates. This observation is not consistent with those in well studied foodborne outbreaks, in which foodhandlers have been found among groups with highest infection rates. Further definition of mechanisms of secondary spread must await results of controlled clinical studies.

Thus far, *S. derby* infections have been considered without regard to the nature of clinical illness. Although described in more detail in a later presentation, certain clinical observations are of epidemiological importance. They are as follows: (1) Among secondary cases, asymptomatic infections occurred at least as often as symptomatic infections, with a frequency proportional to the vigor with which they were sought. (2) Severity of illness varied, but life-threatening or typhoidal illnesses were extremely rare (2.0 percent of symptomatic infections, and 1.0 percent of total infections in one study of 303 cases). (3) Fifty-eight fatalities have occurred among patients harboring *S. derby* at or near time of death. However, nearly all patients who died were suffering life-threatening illness prior to acquiring *S. derby* infection. Thus, individual *S. derby* infections were for the most part a nuisance – expensive, uncomfortable, and inconvenient – but seldom life threatening.

Specific Attempts to Control Secondary Spread of Infection. Attempts to control secondary spread of infection were fraught with many obstacles, and in the final analysis, each may be found wanting. Most clinicians chose to treat all *S. derby* infections with antimicrobial agents. Specific antibiotic therapy appears to have failed to alter both the clinical course of most uncomplicated *S. derby* infections and duration of intestinal excretion of the organism. This impression has not been confirmed by controlled clinical studies, and a retrospective case analysis is not yet complete.

Most time-honored techniques ("intestinal isolation") designed to control enteric infections, primarily typhoid fever, were instituted. Persons known to be infected were isolated and hospital personnel were admonished to observe stringent rules pertaining to personal hygiene. The most rigidly enforced regulation was that requiring thorough handwashing following each patient contact. Infections among employees were actively sought. If found, the employee was prohibited from patient contact until free of the organism. With certain exceptions, these were the basis of attempts to control the spread of infections in most hospitals. As of March 1964, collectively, they have not yet been completely effective in halting secondary spread of infection. The degree to which they may have helped is impossible to assess. Certainly, no increase in rate of appearance of *S. derby* infections occurred during their enforcement. However, one indication of the vigor with which they were pursued may be found in their effect on hospital-acquired staphylococcal infection. In at least two hospitals, the incidence of hospital-acquired staphylococcal infection precipitously declined during periods of rigid enforcement of isolation procedures and handwashing regulations. Clearly, the measures prescribed in two hospitals were followed, at least to a degree sufficient to control the staphylococcal problem.

Current Status of the Problem. During the 11-month period encompassed by this report, 2137 isolations of *S. derby* from humans have been reported to the CDC. By September 13, 1963, when reporting of detailed data pertaining to each *S. derby* isolation was discontinued, 840 hospital-associated cases had been accumulated. Investigation of at least 50 percent of cases reported since that time reveals that the outbreak has remained confined to hospital patients or employees. With very few exceptions, cases continue to originate from those hospitals experiencing secondary spread of infection. One exception is a large teaching hospital in the Midwest that has experienced cases only since August 1963 in a pattern suggesting secondary spread of infection, without occurrence of apparent primary cases prior to that time.

Reasons for the striking increase in reported *S. derby* infections during the past 2-1/2 months vary from hospital-to-hospital and are not always clear. The following have been invoked to explain the increase in one or more hospitals: (1) A previously unrecognized reservoir of asymptomatic excreters of *S. derby* has been uncovered as increased numbers of enteric cultures were performed for other reasons (e.g., epidemic viral gastroenteritis, research projects). (2) Gradual relaxation of control measures has permitted an acceleration in the rate of secondary spread of infection. (3) The common source vehicle of initial infections has been reintroduced. Further studies are in progress.

Comment and Conclusions. An extremely large amount of data has been gathered that pertains to this outbreak. Its sheer volume and the variety of sources of this data pose obvious limitations to its use. In spite of these limitations, certain conclusions are justified. They relate to three general areas: (1) the basic pattern of occurrence of *S. derby* infections, (2) results of attempts to control spread of infection, and (3) nature of the *S. derby* organism. The epidemiology of infections due to *S. derby* followed one of two patterns in most hospitals. In the first, a small number of primary infections occurred, related to a common source; no subsequent infections occurred. The second pattern was one of similar occurrence of primary infections followed by the appearance of a variable number of secondary infections. These secondary infections occurred in a pattern suggesting person-to-person spread of infection. The distinguishing features of hospitals experiencing secondary spread of infection were a greater number of primary cases and the occurrence of these earlier in the course of the epidemic. That secondary infections were not overlooked in those hospitals failing to report new *S. derby* infections is substantiated by negative results of at least two stool culture surveys among patients and employees in such institutions. The occurrence of person-to-person spread of salmonellosis by other than the food-borne route has not been widely acknowledged (7). Study of this outbreak has graphically demonstrated that such spread does in all likelihood occur.

Results of attempts to control secondary spread of *S. derby* infections have been disappointing. Traditional techniques for institutional control of enteric diseases are surely not a panacea. Observations from this outbreak could be interpreted as consistent with the hypothesis that, as practiced, these techniques are totally without value. The need for reevaluation of the effectiveness and indications for such procedures are readily apparent. Some have taken dim view that since these control measures at best only slow the rate of occurrence of new cases, they should be abandoned entirely. Since illness with *S. derby* has not been severe, this approach might be useful or justifiable in a controlled, studied environment; but the dangers of potential buildup of large inocula in a general hospital make complete laissez-faire unthinkable.

Until there is sufficient knowledge of the epidemiology of infections due to other salmonella serotypes, it appears wise to attribute certain specific conclusions derived from study of this outbreak to *S. derby* alone, and not to salmonellae in general. *Salmonella derby* clearly distinguishes itself from most enteropathogenic organisms. It has demonstrated a remarkable ability to persist in both the environment and patient population (7,8). In a study of persistence of *S. derby* infection among institutionalized chronically ill adults, one-third of those initially infected were found to harbor the organism 6 months later. Such ability has seldom been attributed to other salmonella organisms. It is fortuitous that this outbreak was caused by an organism with little capacity to cause severe or life-threatening illness. Perhaps epidemiologic virulence (the ability to spread and persist) is acquired at the expense of tissue virulence (capacity to produce disease). However, until this is proved, we are enjoined to derive all that can be learned from our experience with *S. derby* in institutions. There are over 800 different salmonellae. Perhaps the next will be less a purely *social* butterfly.

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Discussion

DR. LANGMUIR: A number of you have had problems with salmonellosis in your hospitals. Would you like, at this time, to make brief comments concerning your particular problems?

DR. NEWELL: May I ask Dr. Sanders what his conclusions are as to why only hospitals were involved in the *S. derby* interstate outbreak of 1963-64? Were the populations involved more at risk because of the source, because the diagnosis was more readily made, or because these people were more susceptible to disease? There have been a number of other similar, smaller-scale outbreaks in closed institutions, but they have not been restricted to hospitals. They have been in convalescent homes or other closed communities. The interstate outbreak seems rather exceptional because only hospitals were involved.

DR. SANDERS: In my introduction, I also included institutions of the nursing home type for the care of chronically ill and debilitated adults. In one institution, mentally retarded children were involved, so I do not think the *S. derby* outbreak was confined strictly to hospitals.

DR. TAYLOR: May I ask Dr. Sanders if there were any situations where a patient was transferred from one hospital into another hospital where he introduced the infection or was food the source of all the infections?

DR. SANDERS: As you can imagine, this is extremely difficult to document. I can think of one specific instance involving two hospitals, very early in the outbreak, where spread by a patient could have occurred. When we began studying this particular situation, we were turning up so many infections that we could not say with absolute certainty that the transfer of the one patient led to subsequent infections in the second hospital. There was suspicion in one instance, but I do not think this situation occurred more than once.

DR. HART: Did the hospitals that had the infections use cracked eggs to make eggnog?

DR. SANDERS: We investigated this in 15 hospitals. All had purchase orders for grade A medium intact eggs. In at least one instance when these shipments were examined, the order was found to contain large numbers of cracked eggs, many of which were fecally contaminated. I am not trying to assess the blame. Is it the hospital's responsibility for accepting these eggs when they are paying for grade A medium eggs or is it the fault of the wholesaler or the retailer who is shipping eggs to them? I can say certainly that cracked, fecally contaminated eggs did enter some of these hospitals during the study period.

DR. LEVINE: Dr. Sanders and I have discussed this in a lengthy telephone conversation, and we do not agree on what should have been presented to the public with reference to the origin of the primary outbreak of the disease in these hospitals. This is beside the point.

In looking at the broad aspects of the problem, I think that the CDC really should be brought to task for the type of press releases that were sent out to the nation at large and the inferences that the public were asked to make from these releases. These outbreaks were localized in hospitals. The population at large was not endangered nor were the individuals who contracted the infections.

I might say, in passing, that because of these inferences that were drawn without absolute proof, a lot of damage, economic and otherwise, was done to the industry.

DR. LANGMUIR: Dr. Levine, for the benefit of the audience, please identify yourself fully, and let me take the prerogative of allotting 10 minutes to discuss this very broad area now because I do not want to be in the position of in any way closing discussion.

DR. LEVINE: I am Head of the Department of Avian Diseases at the New York State Veterinary College in Ithaca, New York. The reason I am involving myself now in this discussion is because parts of the field investigations were done in New York State. Hospitals in New York State were involved; and, as far as I know, there has not been a single instance of proved infection in eggs in these hospitals.

DR. LANGMUIR: Could you identify now a little more specifically the news release that you object to or are there so many that you cannot identify them? I am sure the discussion will be clearer if you can specify them.

DR. LEVINE: I cannot identify them specifically. There were just two that came to my attention.

The one which I think was the first one that hit the press was a statement which had to do with "it was presumed or it was suspected that eggs were the source of these infections." The second one, which was released much later, enjoined the poulation at large from eating cracked eggs in order to avoid becoming infected with *Salmonella derby*. I asked Dr. Sanders over the phone where I could buy a dozen cracked eggs. I did not know that that was a marketable commodity of any significance.

DR. LANGMUIR: Let me try to keep this to the point and to the issues. The second news release that you identify I am sure is the official release that was issued by the Surgeon General. We prepared the initial draft of it, and Dr. James Goddard, Chief of the Communicable Disease Center, took a personal interest in this and carried it to Washington. It took about 18 hours to get it cleared in Washington through the officials in charge of the Food and Drug Administration. It was taken and discussed at some length with the Assistant Secretary of Agriculture, so that this was not just a fly-by-night release. It was a thoroughly and carefully reviewed statement with all of the facts known.

It was prepared over a period of several days and was released at least a month after most of the basic facts were known. So that I have no thought that we should not stand on and defend it. I want to be sure that the facts are known around the issue we are discussing.

Regarding the earlier release, I am not sure I can identify it. I believe it would be wise for Dr. Hanlon (formerly Director, Community Health Services, Philadelphia Department of Health) to comment in general about this and at least to put a few facts on the line, because there was, I believe, an official release of this to the public in Philadelphia. I want to be sure that this problem is recognized, because we in government face a very difficult and delicate issue whenever a problem reaches the point of sufficient public concern to involve the press. If the newspapers scent headlines while following a trail, then we really have a tough problem to handle. Dr. Hanlon was one of those who was on the firing line in this situation. Would you comment, Dr. Hanlon?

DR. HANLON: To begin with, I would like to second what Dr. Langmuir has said about those of us who are in positions of public responsibility. I stress the word "public." Our responsibility is somewhat different from that of the research worker or of the industrialist and businessman, although these latter come closer to our position.

We must operate with a somewhat awesome and frightening responsibility to the public at large and to all segments of it. Furthermore, we operate very much in a fishbowl. It is practically impossible for those of us who work in public agencies such as health departments to hide anything. We are constantly under surveillance by the public and particularly by those who work in the communications and news field. In view of our national political philosophy, this is as it should be.

There is not a day that goes by that we are not asked about something. Beyond that, there is something very real about passage of information, correct or incorrect, warped or not, among the public at large. And as far as any unusual situation in a hospital is concerned, it does not take very long for the grapevine to start working, both among the patients and among the hospital staff. Beyond this, everybody who is a patient in the hospital has family members and neighbors back home; so, believe me, it is not very long before the word gets out.

In this particular instance, as Dr. Sanders has illustrated and described so well, within a short time very strong presumptive evidence pointed toward eggs. It was only common sense on the part of anyone working with the affected hospitals that action had to be taken to prevent the further introduction and spread of the infection. This, we did. We tried to do it as quietly and as effectively as possible in cooperation with the hospitals concerned.

But even this gets on the grapevine; and, in no time at all, the hospitals were receiving calls from the press, television, and radio. The health department in turn is asked all sorts of questions, sometimes quite embarrassing and even to the extent of accusing officials of hiding things from the public. Under such circumstances, it is only common sense again to try to circumvent misunderstandings by means of a carefully worded statement of the true situation. I would summarize my comment by saying that when one decides to work as a professional in the broad public interest, one cannot duck these issues. One cannot refuse to take whatever steps are necessary, based either upon presumptive or clear-cut evidence, in order to protect the public at large.

DR. LANGMUIR: Let us see if we cannot focus the issue sharply. I believe that the first release that got rather wide distribution was the one from Philadelphia, which admitted the existence of a problem in several hospitals there. As I remember it, Dr. Hanlon, you did recommend that hospitals in your city discontinue the use of raw eggs, did you not?

DR. HANLON: Yes, that is correct.

DR. LANGMUIR: So this is a pretty damaging statement that you made, and you and I are both here under scrutiny for the responsibility and competence with which we did this.

DR. HANLON: On that basis, then, given the responsibility to protect the public at large and segments of it, such as hospital patients, with whatever evidence we have up to a certain point, we must act. Up to the time of the Philadelphia release that was referred to by Dr. Langmuir, we really did not know the extent to which the public at large was or might be affected. Obviously, one cannot afford to wait in the face of a potential community-wide epidemic. One must act. There is no other choice.

DR. LANGMUIR: I would like very much to call on anyone in the room, particularly one of those affected by this publicity, to make any comments or observations they would like to.

DR. ISENBERG: I was not affected economically as far as the egg business is concerned. I am, however, a hospital microbiologist representing one of the involved hospitals. I wonder if the people who are so offended that their industry has been attacked have taken into consideration the costs that the average hospital incurs as a result of such an outbreak.

I think we tested approximately 9,000 stool cultures from employees in order to detect carriers. I am not counting the number of those done on patients and their families. I would estimate that the hospital's cost in days off of personnel that might have salmonellosis or might be suspected simply because they presented with symptoms of diarrhea, in addition to the laboratory expenditure, is well above $15,000 for the year 1963, which for a voluntary hospital is a considerable sum of money.

I mean only to emphasize that there are two sides to economics. I think sometimes, as Dr. M. J. Snyder from the University of Maryland said so very brilliantly, one can never figure out the economics of human disease and, therefore, one disregards it completely.

MISS LALLY: I am with the Institute of American Poultry Industries. At the time this publicity occurred, I happened to be in Virginia and went from there to Washington and then to Philadelphia. We were appreciative of the information we received both from the CDC and from the Philadelphia Health Department as to the facts of the problem, and I would recommend that in cases such as this, one get in touch with the proper authorities and learn the primary facts of the case and the reasons behind the issuing of any statement.

I believe the statement in question was issued by the Public Health Service to counteract some wild rumors that were in the daily press and to put the actual facts of the case before the public.

DR. CARPENTER: Commenting on the two releases—I saw them both and thought they were 95 to 98 percent factual. The things that ruin our industry on an overnight basis are the headlines that our local papers put over the material, and we obviously have little or no control on that point.

I have a series of headlines that I collected during this outbreak. You could have picked up a morning paper, let us say, in Dallas, which might have read: "Public Health Says Dallas Eggs Are Safe to Eat." Headlines in another paper might have read: "Other Eggs Dangerous. Local Eggs Okay." Now these are million dollar headlines, yes, multimillion dollar headlines. I do not think, Dr. Langmuir, that the well intentioned, informed people are fighting factual press releases. They are fighting for their economic lives and what happens to the releases when they get into the hands of the headline hunters.

DR. LANGMUIR: On the publicity angle, we are acutely aware of our responsibilities. We are well aware that we may make slips. We have a large number of people in the field, and all kinds of things may happen. We have an official publication, The Weekly Morbidity and Mortality Report, that goes out once a week to all of the newspapers. It is on file in all the major libraries. It has a circulation now of 10,800 people, mostly official professional people but also newspapers. So this is our main line of communication with the fourth estate. We are responsible for the factual material that appears in this official report.

We are aware that, as of the hour we go to press, the information is correct. It comes to distribution and release on Friday morning, and the situation is such that the data may no longer be correct at the time it reaches the newsmedia. Also very beneficially from our point of view and I think from everybody's point of view, by the time it hits the press, the heat of the event on the Monday, Tuesday, or even Wednesday morning has already gone down immensely unless it is a very major problem. So that we have this protection; this little waiting period, I think, goes quite a long way toward reducing the extent of some of the headlines.

News releases that emanate from Washington create the headlines. It is the little inch or two on the back page where you see information from The Weekly Morbidity and Mortality Report. Dr. Levine, would you care to comment further.

DR. LEVINE: I think, Dr. Langmuir, the essential problem here is that wonderful word in epidemiology, "presumptive." That is a marvelous word, and we hide many sins underneath it. The evidence that was acquired at the time this material became available to newspaper reporters, was presumptive insofar as the origin of the original cases is concerned. Insofar as I can understand, even now, the evidence is still presumptive.

I think it is correct that you have a primary responsibility to the public. On the other hand, you also have the responsibility of correcting wrong inferences or misinterpretations that may be made from releases in various newspapers over the country. I say that when the evidence is presumptive and when people are trying to make something more than "a presumption" out of it, it is also your duty to correct these notions as quickly as possible. This was not done, and this is the sad part of it. The false impression that people get at the beginning is not corrected, even after a retraction is published. If some kind of corrective statement had come from CDC, that would have helped at least to soften the situation. Instead, commissioners of agriculture all over the country had to issue press releases of their own trying to counteract the damage that had been done.

DR. LANGMUIR: Are there others who would like to comment on the question of responsibility and damage done by publicity?

DR. WADE SMITH: The question was asked about the effect on some of the victims of the news release that was so unfortunately blown up. While I was indirectly a victim, I have been working directly with several such direct victims. We tried to see what kind of an effect this announcement had on the poultry industry. Of course this is difficult because of seasonal and annual variation, and variation from other unknown sources. But as nearly as we could estimate, for a period of some 4 to 6 months after this splash, there was probably an egg price depression of somewhere near a cent a dozen to the poultry man.

A cent a dozen does not sound like very much to those of us who buy a dozen eggs a week. But a cent a dozen for 6 months is approximately half a bird's production, that is 10 dozen eggs per bird, 10 cents per bird. I know a man within an hour's drive of where we are right now with 120,000 birds—who, incidentally, ships all of his eggs to Philadelphia; 120,000 birds at 10 cents a bird adds up to $12,000 to one individual.

I know perhaps a dozen such people who each have somewhere between 80,000 to 150,000 birds who paid dearly for the unfortunate publicity caused by the announcement.

The responsibility for the way the announcement was handled is primarily that of the popular, sensationalizing press, that is true. However, it would appear to me that those in a sensitive enough position to make such an announcement might also bear the responsibility of making it in such a way that it does not lend itself to such exploitation. This statement, as nearly as I could have read it to start with, did state the facts clearly and simply as they were available at the time, and I quite agree that you must take action at a certain time and when you must take action you have to use the only information you have at hand. But, at the same time, such a statement could have been made with limitations so that a nonsophisticated readership could not have magnified it that way.

I want to drive that home with one more point. At this conference yesterday afternoon, after having heard something about the magnitude of the salmonella problem in eggs and poultry, I heard one lady say to another, "Does this mean that we should stop eating eggs altogether?" This was heard among an audience that is supposed to be able to understand the salmonella problem and keep it in its own perspective. This news release did not go to a sophisticated audience.

DR. LANGMUIR: Are there others who would like to comment or to talk about this very crucial, rather emotionally charged, but very fundamental question?

MR. TURNBULL: Was there at any time in public health statistics an indication of a higher incidence of S. derby infection among the general public last spring?

DR. LANGMUIR: That is a pretty general question. Dr. Brachman, can you answer this?

DR. BRACHMAN: The slide that I showed yesterday pertaining to our surveillance data indicated that, based upon isolates reported to us from the State laboratories, there has been a general and consistent increase in the number of salmonellae isolated from throughout the country since we began our surveillance program. We think this represents both a seasonal increase and a general increase due to more interest in salmonellosis, so that more cases will be investigated, more cultures obtained, and thus more salmonella isolated. But, I should like to reemphasize the point that Dr. Sanders made this afternoon, that we have not noted any increased recovery of S. derby from nonhospitalized patients during the spring or summer of 1963.

MR. TURNBULL: Then it seems to me this fact should have been made clear to the public that this was a hospital situation and not a general public situation. Had this occurred, I doubt that the public would have become alarmed.

DR. BRACHMAN: The news release that was released to the public from the Public Health Service clearly stated that this was a hospital problem.

DR. LANGMUIR: Since this matter has been brought up as a charge of responsibility, I have proposed that Dr. Goddard tell us the steps that were taken when it became apparent to us that a news release from the Surgeon General was imperative. A brief summary of the facts leading up to the preparation of the release may be in order. The situation in Philadelphia was that several hospitals became involved and the health officer in Philadelphia felt constrained to make a recommendation to the local hospitals. This hit the Philadelphia papers and was picked up somewhat more broadly. Also, in the Morbidity and Mortality Weekly Reports the facts of a S. derby problem in hospitals was reported week by week although the attribution to eggs was not made in any of the early releases. We were aware that this was coming and over a period of some weeks I kept briefing Dr. Goddard of the problem. When the situation reached the point in early July that a release was going to be necessary simply because reporters in Washington were demanding statements from the Surgeon General, he was obligated to make some sort of reply. We worked on it here, particularly Dr. Goddard and I, drafting and redrafting statements. Dr. Goddard, would you please continue from this point.

DR. GODDARD: I noted in the second paragraph of the Surgeon General's release that the statement was made, "It was felt that an epidemic of hospital-associated salmonella disease was occurring and an investigation was launched by the hospital, city, and State and 17 CDC personnel." Then it went on to tell the number of cases and so forth. Now we obviously feel that we are in a position where we must act in a very responsible fashion in this type of episode as in all others.

The Surgeon General is charged by law with the job of protecting the health of the American public; and, as indicated, queries were being directed to his office. The information that was then available as the result of field studies which were carried out as judiciously as possible were reviewed by Dr. Langmuir with me. I then took this information plus a suggested form of a news release to Washington and reviewed it with the Surgeon General. He asked his public affairs man to review it, and several changes were suggested.

We attempt to be as fair and accurate in reporting facts as we possibly can. I speak as an administrative person in this matter rather than as a scientist. I have to review what the scientist has done and make certain that he is indeed impartial. Sometimes, since they are human, their biases often begin to show because they have invested a lot of time and effort in a problem. We try to make sure that the scientists remain impartial and that the facts are laid out. Then, it is our obligation to present the facts to the public and make recommendations based on these facts. Unfortunately, there are some newspaper reporters who will present the data in a sensational way in order to catch the reader's eye, but these men are in the minority. Most of the reporters that we encounter are responsible individuals.

Admittedly, the public is not always capable of proper interpretation of these facts, but it is then our responsibility to objectively place the data in its proper perspective. At no time did we state eggs should not be eaten. We did indicate that only grade A eggs should be purchased and only well-cooked eggs should be served to infants and the elderly in the family—the same recommendations as made for hospital patients.

In this particular situation, we feel that we discharged our obligations to the best of our knowledge and abilities. I hope this discussion has cleared up some of the misconceptions that inadvertently developed during the publicity given to the Salmonella derby investigations.

The Field Studies

John R. Boring III, Ph.D.[1]

An analysis of the *Salmonella derby* outbreak is especially pertinent because this epidemic is representative of a commonly occurring problem which could pass unrecognized if it were not for extensive surveillance. Similar large but unsolved outbreaks were caused by *S. reading* in 1956 and *S. hartford* in 1961. A distinguishing feature of all these outbreaks has been the almost simultaneous increase in isolations of one salmonella serotype in scattered but contiguous locations. The recognition of such epidemics comes only when the increases in isolations are considered together. This feature is enough to suggest a common source in each epidemic, most probably a food item of wide distribution. The repeated occurrence of these outbreaks affords an opportunity for investigation which could uncover a general pathway by which salmonellae are introduced into human populations.

FIELD STUDIES

The starting point in the *S. derby* investigation was the observation that the majority of patients with initial hospital-associated cases had consumed raw or undercooked eggs. This prompted an examination of eggs in some of the hospitals involved and led to a bacteriological investigation of the eggs, chickens, and feeds on or from the farms supplying the hospitals.

Salmonellae were not isolated from 1,870 Grade A eggs collected from 3 of the involved Philadelphia hospitals 1 month after the epidemic was recognized (Table 1). An attempt was then made to trace the eggs back to their farm sources, based on the possibility that there was residual contamination or unrecognized flock infections still existing. Because of the nature of egg distribution, tracing the eggs presented a problem. Approximately 12,000 farms could have supplied eggs to one or more of the 15 hospitals initially involved. The solution was to examine eggs collected by the two distributors who were the only ones that could have supplied eggs to all 15 of the hospitals. One distributor was located in Pennsylvania and one in New York. A sample of eggs from the Pennsylvania distributor was examined about 2 months after the epidemic was recognized. *Salmonella derby* and *S. heidelberg* were the only types isolated, and they were from cracked or "checked" eggs (Table 1). It should be mentioned that *S. heidelberg* was isolated only after repeated subculture. In all, 4,110 Grade A and B eggs from several hospitals and the egg distributor were examined without detecting salmonellae.

The next step involved examination of 33 farms which along with others supplied eggs to the distributor in Pennsylvania from whose "checked" eggs *S. derby* had been isolated. Pooled chicken droppings and mixed feeds were collected from these farms approximately 2 months after the epidemic was recognized. Salmonellae were isolated from chicken droppings on 4 farms (Table 2). The percentage of salmonella recoveries was low and *S. derby* was not isolated. The isolation of *S. binza* at Farm W assumes some importance to be mentioned later.

Eight of the 33 feed samples collected at these farms contained salmonellae and one from Farm W harbored both *S. derby* and *S. montevideo* (Table 3). *Salmonella manila*, an infrequently isolated type, and *S. binza* were also isolated from feed samples taken from other farms in the area.

One large mill provided feed ingredients for Farm W and many other farms in the area under investigation. Samples from this mill were collected and examined approximately 3 months after the epidemic began. Salmonellae were isolated from fish meal and rendered meat products, both ingredients of chicken feeds (Table 4). *Salmonella derby*, *S. montevideo*, *S. cerro*, and *S. worthington* were isolated from one of two rendered products and *S. thompson* from fish meal.

The mill supplying these feed ingredients was in turn supplied with products from three fish meal plants and six rendering plants. Samples from the fish meal plants and four of the rendering plants were collected approximately 3 months after recognition of the outbreak. Environmental swabs as well as finished product from the fish meal plants contained numerous salmonellae but no *S. derby*. On the other hand examination of the environment and products of the rendering plants revealed a high degree of salmonella contamination (Table 5).

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Table 1.–*Salmonellae isolated from eggs*

Source and grade	Number of cultured eggs	Number of cultured egg pools[1]	Pools positive	Serotypes isolated
Hospitals involved:[2]				
A.	1,870	104	0	
Distributor (collected on farms):[3]				
A.	2,120	117	0	
B.	120	8	0	
"Checked" and cracked	330	22	5	*derby* (3 pools) *heidelberg* (1 pool) *derby* and *heidelberg* (1 pool)

[1] Eggs cultured in pools of 15 to 18.

[2] Epidemic recognized in April 1963; hospital eggs collected May 30, 1963.

[3] Farm eggs collected June 20 and July 6, 1963.

Table 2.–*Salmonellae isolated from chicken droppings from 33 farms, (total specimens–1,754)*[1]

Farm	Serotypes recovered	Number of isolations
W	*binza*	1
Z	*infantis*	35
A	*typhi-murium*	1
X	Not identified (4, 5, 12: 1, 2 monophasic).	1

[1] Epidemic recognized in April 1963; specimens collected in June 1963.

Table 3.–*Salmonellae isolated from feed samples obtained at 33 chicken farms, compared to other isolations from same farms*[1]

Farm	Serotypes recovered		
	Feed	Chicken Droppings	Environment
W	*derby* *montevideo*	*binza*	*anatum.*
Z	*manila*	*infantis*	
X	*senftenberg*	Not identified (4, 5, 12: 1, 2 monophasic).	
P	manila		
L	*senftenberg*		
M	*montevideo*		
K	*binza*		
B	*kentucky*		

[1] Epidemic recognized in April 1963; samples collected in June 1963, one sample from each farm.

Table 4.–*Salmonellae isolated from feed ingredients*

Type of specimen	Positive samples	Total samples[1]	Serotypes isolated
Alfalfa pellets	0	1	
Soybean meal	0	1	
Wheat meal	0	1	
Vitamin supplements	0	9	
Fish meal	1	1	*thompson.*
Rendered products (meat scraps).	1	2	*derby.* *montevideo.* *cerro.* *worthington.*

[1] Epidemic recognized in April 1963; specimens of feed ingredients collected on August 9, 1963.

Table 5.–*Salmonellae isolated from rendering plants supplying wholesaler*

Plant	Number of samples	Percent positive	Number of samples with *S. derby*		Frequency of serotypes (all plants)[2]
			Number	Percent[1]	
A.	34	97	18	53	*derby* (44).
B.	35	83	11	31	*senftenberg* (25). *montevideo* (23).
C.	31	74	11	35	*anatum* (22).
D.	27	66	5	19	28 additional serotypes isolated less than 20 times.[3]

[1] Percent of total samples.

[2] Figures in parentheses denote number of isolations.

[3] Including *S. binza* (6); *S. manila* (2); *S. heidelberg* (4); *S. infantis* (2).

Salmonella derby was isolated from all plants and S. montevideo from three. The percentage of samples containing S. derby ranged from a low of 19 percent in one plant to a high of 53 percent in another. Salmonella derby was the most frequently isolated type, occurring almost twice as often as the second most frequent type, S. senftenberg. Salmonella montevideo and S. anatum were also commonly found. Included among the many other serotypes isolated were S. manila and S. binza, previously found in the finished feed (S. binza was also isolated from chickens at Farm W), and S. heidelberg, which was one of the types isolated from "checked" eggs.

A sample of 360 "checked" eggs from the New York distributor who was the only other distributor who could have supplied all 15 hospitals initially involved, was examined. Salmonella typhi-murium was isolated from one slurry (18 eggs per slurry). Nineteen farms supplying this distributor were then visited and chicken droppings and feed were collected. Chickens on 12 of the farms were positive for salmonellae. Salmonella thompson, S. kentucky, S. typhi-murium, S. infantis, S. binza, and S. saint-paul were the predominate types in the chickens. Several feeds were positive and S. binza, S. saint-paul, S. montevideo, S. cerro, and S. senftenberg were the types isolated. Since S. derby was not isolated no attempt was made to trace the feed to its source.

DISCUSSION

These observations and especially the isolation of S. derby from 5 of 22 slurries of cracked and "checked" eggs support the epidemiological evidence that contaminated eggs were a possible source of this epidemic. Whether the organisms were initially introduced into the hospitals in cracked eggs is not known. However, shortly after the epidemic was recognized, it was observed that cracked and dirty eggs were being used in at least one of the hospitals involved. Particularly germaine to the failure to isolate salmonellae from Grade A eggs is a study done by Philbrook and his co-workers (1) in 1961. They described an outbreak of S. typhi-murium in a mental hospital which was traced epidemiologically to eggnog. This institution was supplied with eggs from its own farm, and birds infected with S. typhi-murium were separated from the main flock and their eggs examined for the organism. Of 1,117 eggs produced by 29 birds infected with S. typhi-murium, only 3 were found to contain this organism. Thus a rather small number of eggs may become contaminated even when they are laid by animals known to be harboring salmonellae. This observation makes even more pertinent the isolation of S. derby from "checked" eggs in the present study.

The results of the attempts to determine the original source of the S. derby organisms also indicate a pathway by which this or other salmonella organisms may be introduced into human populations. This pathway and its significance have been suspected for some time especially by investigators in Germany and England, and it was believed but never firmly shown to be the way in which S. reading was introduced into humans. Several studies have shown that salmonella serotypes may be isolated from fish meal, rendered meat, animal feeds, poultry, and less commonly from eggs. The difficulty has been, and to a degree still is, in connecting these isolated findings in a manner that will persuasively show the sequential passage of a salmonella serotype from feed ingredients, to feed, to poultry, and eventually to man. Newell and his co-workers (2) did much to indicate such a chain in their study of salmonellosis in Northern Ireland. On one farm, they isolated S. infantis from pigs, from hen eggs, and from pig meal which was distributed to the pigs in the same bucket as that used for the poultry feed. Tracing the pig meal back to its source, they then isolated S. infantis from Angolian fish meal, one of the pig meal ingredients. Again, the inference from these studies is that man may be infected by infected fowl and that the latter may be infected from ingesting contaminated feed.

In the present retrospective study, the chain of infection was not as clear. A pattern of distribution was established whereby it could be shown that farms which supplied eggs to the involved hospitals received feed from a source later found to be contaminated. The isolation of S. derby as the predominant serotype in rendered products and in eggs, and its presence in feeds was further supportive evidence for this pathway of infection. The prevalence of S. derby should also be noted. In recent national surveys, it has been ranked as the 11th most common isolate from chickens and the 13th most common from animal by-products and rations. There has been but one reported isolation from whole eggs in this country and that was by Watt (3) in 1945.

While the evidence that salmonellae may be passed from feed ingredients, to feeds, to animals, and eventually to man is still presumptive, the potential for this pathway is clear. One lesson from the S. derby outbreak that appears applicable has perhaps been stated best by Newell (2) in speaking of salmonellae: "These organisms will continue to be passed to humans unless it is recognized that the only effective place to break the chain of infection is by attempting to break the animal cycle. This is possible in some instances by the bacteriological control of animal feeding stuffs, and should be the concern of both health and veterinary authorities."

While this evidence does not conclusively establish a chain of infection in the S. derby outbreak, it does, however, indicate a potential pathway whereby salmonellae may be passed from feed ingredients to poultry or their products and eventually to man.

SUMMARY

Investigations were undertaken to determine whether eggs were the source of infection in the S. derby epidemic, as was indicated by epidemiological observations.

Salmonella derby was isolated from:

1. Cracked and checked eggs obtained from a distributor supplying all of the hospitals initially involved.
2. Feed from 1 of 33 farms supplying the egg distributor.
3. Rendered products obtained from a feed wholesaler.
4. All rendering plants supplying the feed wholesaler.

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Role of the City Health Department

John J. Hanlon, M.D.[1] *and*

Lewis D. Polk, M.D.[2]

A local health department confronted with an outbreak of communicable disease, such as an outbreak of Salmonella derby gastroenteritis, has a number of complex responsibilities to many segments of the community affected. Obviously, it first has a special responsibility to assist those institutions already involved, in their efforts to establish and effectuate adequate intramural control programs. It also has a similar duty toward other institutions not yet involved in the outbreak but which potentially might become affected. It has, of course, certain legal and moral responsibilities to the public and to the government of its community, of its State, and frequently of the nation. In the 1963 S. derby outbreak in Philadelphia, these agencies included the Pennsylvania Department of Health, the Pennsylvania Department of Agriculture, the Communicable Disease Center of the U. S. Public Health Service, the U. S. Food and Drug Administration, and the U. S. Department of Agriculture. Beyond these there is, of course, the responsibility to handle the situation in a manner which will result in the least disruption of the social and economic life of the community.

In the spring of 1963, when the outbreak of hospital-acquired S. derby infections became apparent, the Community Health Services component of the Philadelphia Department of Public Health was fortunate in having previously prepared a plan of operations for special situations of this nature. In a series of documents entitled Program Phase Plans, the goals, activities, standards, and procedures were specified for the various existing and potential health program and problem areas for which the agency is responsible. As part of this preplanning, therefore, a blueprint already existed which detailed the respective roles and responsibilities of the various individuals and units in the Community Health Services, should there occur an outbreak of infectious disease which would affect one or more hospitals or similar institutions. As soon as it became obvious that a number of hospitals in Philadelphia were affected by S. derby, this organizational mechanism made it possible for all official activities in relation to these institutions to be directed and coordinated promptly from a pro tem central point within the health department, designated as the Salmonellosis Surveillance Unit.

The responsibility to keep the general public informed, took the form of centralized preparation of news releases to the various channels of community information — the press, radio, and television. Special procedures were quickly devised to handle a large number of telephone calls. The telephone operators at the central switchboard in City Hall were given a list of questions that it was anticipated the public would ask, and the answers which the operators could give immediately without involving anyone else. If the call came from a member of the public and involved a topic not covered in the list of questions and answers, the call was to be routed from the central switchboard to a specified series of telephones in the central Salmonellosis Surveillance Unit. Calls from institutions or physicians, on the other hand, were all routed to another series of telephones in the central Salmonellosis Surveillance Unit. All other units in the health department were promptly provided with background information and advice on how to handle calls relating to the outbreak and to whom to refer calls.

In relation to the mass newsmedia, the health department was soon confronted with the special problem of preparing releases concerning the consumption of eggs by the general public. This became necessary when the public learned of the confidential warnings to institutions that eggs might be implicated as a vector of S. derby. It also was felt necessary to give the public accurate information with regard to what was happening in the local hospitals, in order to counteract numerous rumors and to reassure the public that hospitals were generally safe.

Another area of responsibility concerned all those institutions in the community that might become affected even though they were not yet known to have patients or personnel infected with S. derby. The first step was the telephone notification of all local hospitals of the potential danger of serving raw or undercooked eggs especially to patients who were chronically ill, debilitated, or scheduled for certain types of surgery. Next, telegrams were sent which outlined the actions to be taken in order to prevent or minimize person-to-person transmission, as well as the procedures necessary for surveillance and follow-up of problems as they might be discovered. These telegrams were sent to all general and special hospitals, nursing homes, homes for the aged, and children's residential institutions in Philadelphia. While the use of telegrams was costly, it was done for purposes of emphasis as well as speed. The response and results amply justified the procedure. The next action took the form of visits by multidiscipline teams to each of the dozens of general hospitals in the community that had not yet become involved in the salmonella outbreak. Typically, a team consisted of a public health trained physician as a team leader, a sanitarian, and one or two public health nurses. The sanitarians and nurses of the multidiscipline teams were usually of supervisory level. The team conferred with such key hospital personnel as the administrator, medical director, and directors of nursing, food services, housekeeping and maintenance staff. Topics discussed included the situation in general and in Philadelphia, the importance of reviewing and tightening existing procedures in relation to institutional food handling, patient care, and housekeeping, as well as what surveillance procedures should be set up, and what actions should be taken if the infection invaded the institution.

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As mentioned earlier, the institutions that were already known to be affected by the outbreak received the first and greatest attention. Special surveillance and control teams were developed from the staff of the Community Health Services and assigned to function full-time within each of the affected institutions. Although at least 12 Philadelphia institutions eventually had cases of S. derby gastroenteritis, which were considered to be hospital-acquired, not more than 5 such teams were in operation at any one time. Nevertheless, this type of control procedure placed a severe strain upon the ongoing activities of the agency.

It is important to indicate that the job of the teams was not to sit at a desk in the hospital and merely to coordinate an exchange of information. Rather it was to move continuously throughout all parts of the institution in order to determine, if possible, whatever practices or techniques might be contributing to the introduction and spread of S. derby infection in the hospital. However, upon noting a problem or action needed, they took no direct remedial action themselves. Instead, they advised the administrator of the institution, and assisted him and his infections committee, if such existed, in correcting the problem and in observing the situation after an appropriate interval in order to assure its correction.

It was considered important to have a clearly defined relationship between one individual from the Community Health Services at each institution, specifically the team captain, and one individual designated by the hospital, usually the administrator or someone on his staff, to exchange information. This was necessary in order to avoid the confusion, or at least duplication of effort, that would occur if many individuals from the official health agency and many individuals from the hospital were all transmitting information. Similarly, the team leaders communicated with one individual, the Director of the Central Salmonellosis Surveillance Unit, at the Community Health Services headquarters.

Items considered to be important for the team members to observe consisted of factors that might affect patients or personnel, whether or not these individuals were known to have yet become infected with S. derby. They included medical and nursing techniques, such as handwashing and isolation techniques; food preparation, distribution and service; and other environmental matters, such as laundry procedures, and the handling and sterilization of bedpans. A specific example of an important factor to study was the role of individuals who moved from room to room and who might spread infection from person to person. Such possible transmitters were not limited to the obvious categories of staff, patients, and visitors. Others to be considered included newspaper vendors, outside food vendors, and even the clergy.

It was felt that, in hospitals experiencing a major outbreak of S. derby infections, the surveillance and control teams from the Community Health Services could assist in culturing specimens from individuals with signs or symptoms of infection, and, under certain conditions, assist in general bacteriological surveys of groups of patients and/or institutional personnel. The procedures for handling such individuals found positive by culture were also supervised by the Community Health Services teams stationed in the institutions.

As previously stated, the central Salmonellosis Surveillance Unit in the Community Health Services was originally formed from the nucleus of the specific individuals designated in the organization's Program Phase Plans. In the circumstance under present discussion, it soon developed into the coordinating unit for all of the surveillance and control teams in the institutions throughout the city. The central unit also directed and coordinated the activities of the informational teams which visited hospitals not yet involved. The chief of the central Salmonellosis Surveillance Unit, under the guidance and supervision of the Director of Community Health Services, decided which hospitals should be sent a visiting team, which should have a team in residence, and the composition of teams.

In like manner, the decision as to when a team should be removed from a particular institution was made by the chief of the central Salmonellosis Surveillance Unit, again with the guidance of the Director of Community Health Services. Factors that were considered in making this rather difficult decision were as follows: a significant decline in the number of new individuals found positive for S. derby in the institution, a demonstrated capacity on the part of the institutions's staff to handle its own intramural surveillance and control activities, and the need for team members elsewhere. A team was never withdrawn abruptly from a hospital but was "phased out" in a gradual manner, by having the full team not visit the hospital for 1 day, and then for 2 days, and so on.

Various techniques were used to maintain communications and to provide coordination of operations. At the height of the outbreak, evening meetings were held almost daily involving the Director of Community Health Services, the chief of the Salmonellosis Surveillance Unit, the leaders of the surveillance and control teams, Communicable Disease Center personnel, and key hospital personnel. On several occasions, representatives of the State Health Department and of various Federal agencies other than the Communicable Disease Center were present in Philadelphia and participated in these meetings. On some occasions, it was helpful to address the entire medical staff of an institution to discuss the importance of their cooperation in the surveillance and control procedures.

One of the most useful activities of the central Salmonellosis Surveillance Unit was to prepare daily reports. These consisted of a summary table showing the number of individuals with positive cultures for Salmonella group B at each hospital, with data for patients and personnel indicated separately. In order to obtain the most rapid and up-to-date picture of the current situation, it was decided to use data on those found to be positive for Salmonella group B, even though the organism had not yet been specifically identified as S. derby. This information was transmitted to the central Salmonella Surveillance Unit by the institutional team leaders at the end of each day. It was considered helpful, as the situation developed, to be able to relate the number of individuals with positive cultures at each hospital to some denominator as an indication of the magnitude of that institution's problem. This was done by indicating in the same summary table the actual number of patients or the average daily census, and the total number of personnel normally employed in each hospital. Later, as time passed and patients who early in the outbreak had been found to be positive were discharged, it was felt important to have some measure of the size of the problem still remaining in the hospital. This was done merely by stating the number of patients with positive cultures that were still in each hospital. In other words, both incidence and prevalence data were accumulated for each institution involved.

The above system was needed as a supplement to, though not a substitute for, the usual routine mandated reporting of infectious disease cases to the Section of Communicable Disease Control of the Community Health Services. It was found that the routine reporting system was too slow and did not provide information sufficiently up-to-date for prompt control activities. The reason for this was that the Communicable Disease Control Section necessarily had to wait for the legal and official reporting of cases with all of the detailed specific information for each case before it could list the individual as an officially reported case. For purposes of emergency surveillance and control, a local health department requires a very rapid method of obtaining and maintaining a current picture of the nature and magnitude of the problem in each affected hospital, even if the specific numbers may be slightly incorrect or the data incomplete, and adjustments have to be made later.

Similarly, some clue is needed as to the deaths that occur as part of an outbreak of this nature and which might be attributed to it. Actually, this presented a very difficult problem. Ordinarily, the only circumstances under which a death would be listed in the official communicable disease reports would be those which indicated S. derby as the cause of death on the death certificate. In this epidemic episode, there were none of these. However, it was felt that there should be some information, even if only for later investigation, on all those patients whose death might be attributed in toto or in part to this cause. Therefore, the daily report included the number of deaths at each institution of individuals who at any time prior to death or at postmortem had had a culture positive for S. derby.

From the foregoing, it can be seen that the daily report consisted of several parts: a summary table with numbers of "cases," a brief narrative of highlights, and detailed reports for all affected institutions. The daily report turned out to be an excellent method of keeping surveillance team captains, the Director of the Community Health Services, the Community Health Services Laboratory, other key individuals in the Community Health Services, the Health Commissioner and, through him, top officials elsewhere in the city government, and the communicable disease unit of the State Department of Health up-to-date on the entire situation.

Much of the above is related to intrainstitutional spread of infection. In an attempt to prevent interinstitutional transmission, the central Salmonellosis Surveillance Unit also tried to prevent, to the maximum extent possible, the transfer of a patient with a positive culture from one hospital to another hospital or to a nursing home (either directly or after a short stay at home). With regard to intrahospital spread, it was considered important to be alert to any situation in which a patient might be readmitted to the same or to a different hospital after recent discharge, with an attempt to hide or ignore the fact that the patient had a positive stool culture and therefore needed isolation upon admission and other precautions.

The laboratory necessarily played a very important role in the management of the *S. derby* outbreak in the City of Philadelphia. We did not want to keep a patient in isolation in the hospital or to prevent his discharge home or to a nursing care facility any longer than necessary. Likewise, we did not wish unnecessarily to prevent the return to work of hospital staff found to be positive for salmonella. It was important, therefore, to determine as rapidly as possible whether a given culture from such individuals was positive or negative. This made it necessary to obtain additional personnel and facilities for the processing of unusually large numbers of cultures. Personnel of the Communicable Disease Center of the U. S. Public Health Service instructed local personnel in the rectal swab technique for screening cultures, and in a practical "short-cut" laboratory technique that could rapidly identify salmonella.

In addition, the Communicable Disease Center provided sera for the specific typing of salmonella cultures. This was particularly helpful since, until this time, we had to depend upon either our own laboratory or that of the Philadelphia General Hospital in order to isolate a salmonella organism and to identify it as group B. It was then necessary to send the culture to the State Bacteriology Laboratory for typing in order to determine whether or not the organism was *S. derby*. Under ordinary circumstances, this system was adequate, but in the face of a fast-moving and large-scale outbreak, the routine procedure was too slow for effective action. With the use of the procedures indicated by the Communicable Disease Center, when all aspects of the operation were working optimally, it was possible to obtain serotypic identification of a recovered organism within 48 hours. In order to do this, a special bacteriology laboratory was organized at the hospital that had the largest number of reported "cases." This temporary laboratory was staffed with personnel from both our own Community Health Services Laboratory and the general laboratory of the hospital. Vast amounts of culture media and materials were prepared on short notice. The central Salmonellosis Surveillance Unit coordinated the pickup, transportation, and delivery. This assured that material promptly reached all hospitals that were or might be involved in the outbreak and that specimens would be obtained and sent back to the special hospital-based laboratory for processing.

As has been discussed in other papers, the majority of the activities required for the general epidemiology and over-all surveillance were performed by the Epidemic Intelligence officers of the Communicable Disease Center.

At the same time that all the work described was carried on within institutions, much was also transpiring elsewhere in the community. The Milk and Food Section and many other units of the Community Health Services of the Philadelphia Department of Public Health as well as of many Federal and State agencies participated in the investigation of turkey rolls, eggs, and other products as possible sources of the *S. derby* outbreak in the institutions. This, however, is a major story in itself and could well be the subject of another paper.

SUMMARY

From the foregoing, it is evident that the role of an official local health agency in an outbreak of this nature is indeed complex and varied. One of the most important in this instance was the formation and use of special multidiscipline surveillance and control teams assigned full-time to hospitals significantly involved in the outbreak. Another was the establishment within the health agency of a central over-all surveillance unit to coordinate and oversee the entire operation from the local health department's standpoint. Also important was meeting the public relations and public information needs of the community by the preparation of daily reports on the current over-all situation in the city and at each affected institution. These reports kept all individuals and agencies who needed to have this information up-to-date. A vital factor in the success of the whole operation was the availability of laboratory facilities for the preparation and processing of material so that all patients, personnel, and staff could be cultured and recultured as often as was necessary in order to determine who was infected and what the progress of the epidemic was. This was essential in order to have any reasonable chance of breaking the chain of transmission from one person to another or from one institution to another. The assistance provided by the Communicable Disease Center in information, advice, materials, and personnel was extensive, invaluable, and deeply appreciated. The local health department did make great efforts; but, without the help of the CDC, much that was accomplished could not have been done at all, and much that we still might have been able to accomplish alone could not have been done as quickly or as well.

Finally, it is our opinion that this unfortunate and difficult occurrence provided an excellent opportunity to demonstrate clearly the methods and values of sincere cooperation among health agencies of different jurisdictions and on the several levels of government, among governmental agencies with different primary substantive interests, and between public agencies and private institutions. Despite the inevitable operational and jurisdictional headaches and occasional minor differences of opinion, we firmly believe that all of us are professionally the better for it, and that we have a type and level of respect for each other superior to that which existed before. This was the silver lining of the cloud.

Clinical and Epidemiological Studies of *Salmonella derby* Infections in a General Hospital

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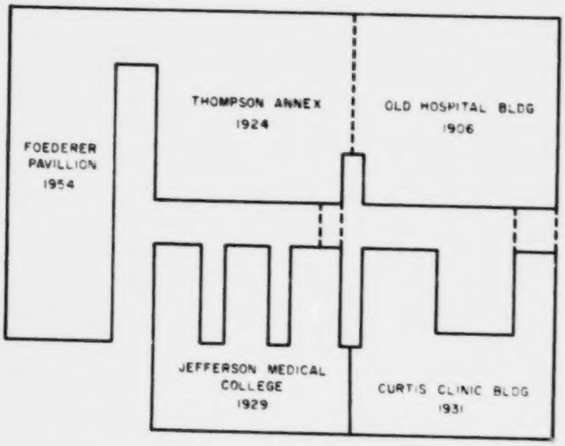
The purpose of this report is to describe an outbreak of hospital-acquired Salmonella derby infections occurring in a large university-affiliated institution. The epidemic had its onset in March 1963 and continues to the present time (March 1964).

Recognized salmonella infections usually result from the ingestion of food which is heavily contaminated with organisms. This process ordinarily results in a fulminating gastroenteritis with an incubation period of 6 to 24 hours. The majority of the individuals involved become ill within a few hours of one another, with a few secondary cases occurring over the next several days or weeks. Hospital-acquired outbreaks of salmonellosis, however, may display an entirely different pattern and may resemble the epidemiological picture of staphylococcal, streptococcal, or Gram negative rod infections. Rubbo and Muskin reported such an outbreak due to S. derby on a hospital pediatric service (1, 2). Salmonella tennessee and S. typhimurium have also been implicated in hospital-acquired outbreaks of infection (3, 4, 5).

Jefferson Medical College and Hospital is a large 700-bed urban institution which is divided into private and semi-private rooms as well as some large open wards. The institutional complex encompasses a full city block and is made up of five inter-connecting buildings of varying ages (Figure 1). All food is prepared in a main kitchen, except for items which are brought occasionally to the patients from outside sources. Personnel within the institution include some 300 members of the attending staff, 175 members of the house staff, 250 staff nurses, and over 1,800 members of other nursing, laboratory, housekeeping, clerical, and maintenance groups. In addition, the medical and nursing students are in close contact with the hospital environment during their training periods.

Figure 1.–*Jefferson Medical College Hospital Philadelphia, Pennsylvania*



Prior to April 1963, the serotype identification of a salmonella species at this institution required approximately 3 weeks. The organisms were isolated and serologically grouped in the diagnostic laboratory and then sent to the Commonwealth of Pennsylvania laboratory for serotyping. This caused little difficulty during previous years because the number of isolations of salmonella was small. During the 7-year period prior to 1963, 104 salmonella isolations were made with 25 being the largest number isolated in any one year. The most common serotype identified during this time was S. typhi-murium (Table 1). Salmonella derby was isolated only once, in July 1959.

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Table 1.–*Patients from whom salmonellae were isolated, Jefferson Medical College Hospital Jan. 1956–Feb. 1963*

[Total isolations 104][1]

Year	Number of patients	Year	Number of patients
1956	6	1960	21
1957	11	1961	25
1958	4	1962	12
1959	[2]21	1963 (Jan. & Feb.)	4

[1] *S. typhi-murium = 26*

[2] *S. derby = 1*

INTRODUCTION OF THE ORGANISM

The usual number of salmonella isolations were made during January and February 1963, that being two each month. In February, a child was admitted to the pediatric service for gastroenteritis and was found to be positive for S. typhi-murium. Shortly thereafter, a number of patients on the same floor acquired diarrhea, but cultures of the patients, nurses, and other personnel failed to reveal the presence of any other salmonella organisms. This outbreak was thought to be due to a viral gastroenteritis. The number of salmonella isolations climbed precipitously in March to 17, 14 of which were group B (Table 2). The epidemiological pattern was confused at this time by a number of factors: first, the delay in serotyping; second, the hospitalization in March of four patients who were admitted with violent gastroenteritis and were positive for salmonella; third, the suspicion that patients were contracting diarrhea due to salmonella during their hospitalization; and fourth, the recognition of two cases of diarrhea due to S. muenchen in the newborn nursery. A cultural survey of all mothers, infants, and floor personnel on the obstetrical floor revealed only the presence of a salmonella E and a D in two of the employees. Particularly noteworthy is the fact that no isolates of S. derby were made during the February or March survey, suggesting that the organism was not present or at least was not widespread in the institution at that time.

The serotyping results indicated that there were two kinds of salmonella disease in the hospital, namely, that due to S. derby which appeared to be originating within the hospital and that due to other salmonellae which were coming to the hospital from outside sources. It also became apparent that the majority of patients contracting S. derby disease in the early stages of the epidemic were suffering from primary gastrointestinal or malignant disease. Attention was focused on the dietary department in an attempt to locate the primary source of the organism. It was felt that the infection was probably food borne, and cultural surveys of the dietary personnel were performed on two occasions during the month of April. No employees were found to be positive for S. derby, but four individuals harbored other salmonella serotypes. Environmental cultures

Table 2.–*Salmonella isolations–Jefferson Medical College Hospital, Mar. and Apr. 1963*

Date	Classification	Location	Salmonella Group	Serotype
1963				
Mar. 6	Patient	[1]52	B	*derby.*
13	Patient	955	B	*derby.*
18	Patient	1264	B	*derby.*
19	Patient	926	B	*derby.*
21	Student Nurse	[2]O.P.	B	*derby.*
23	Newborn	1023	C_2	*muenchen.*
23	Newborn	1023	C_2	*muenchen.*
25	Technician, Tenth Floor	O.P.	E	*meleagridis.*
23	Staff, Tenth Floor	O.P.	D	*enteritidis.*
23	Patient	1056	B	*derby.*
26	Patient	1119	B	*typhi-murium* var. *copenhagen.*
25	Patient	718	B	*typhi-murium.*
25	Patient	1226	B	*typhi-murium.*
26	Graduate student	[2]O.P.	B	*typhi-murium.*
27	Patient	930	B	*derby.*
31	Patient	[1]54	B	*derby.*
Apr. 1	Patient	929	B	*derby.*
3	Patient	967	B	*derby.*
3	Resident physician (anesthesia)	[2]O.P.	B	*derby.*
6	Patient	957	B	*derby.*
9	Patient	721	B	*derby.*

[1] Star Pavilion

[2] Outpatient department

of prepared and unprepared food, ice, water, kitchen utensils, chopping blocks, floors, serving carts, and other items were also negative for salmonella organisms. It appeared from both the epidemiological nature of the outbreak and from the surveys that the epidemic was not arising from within the dietary department at the time the surveys were performed. Ironically, the Chief Cook's stool was negative for salmonella on both surveys, but shortly thereafter he was admitted to the hospital for gastrointestinal surgery and contracted the disease during the second week of his illness.

During the month of April, the laboratory reported 18 new isolates of S. derby. It then became evident that an epidemic was in progress within the hospital and that there was secondary spread of the infection, presumably person-to-person. The help of the local, State, and Federal health authorities was obtained at this time; and intensive patient, personnel, and environmental surveys were conducted in an attempt to determine the mechanism(s) of secondary spread and to continue the search for the source of primary infection. Studies concerning the initial source of infection are the subject of a previous communication and will not be reported in detail here (6). From these studies, it was evident that an extensive outbreak of S. derby infection, presumably involving a common source, was occurring in many hospitals throughout the northeastern United States. Accumulated epidemiologic and bacteriologic evidence suggested that the source of this infection was raw or undercooked eggs, one of the staples in many hospital diets.

THE CLINICAL SYNDROME

The entire clinical spectrum of disease was encompassed by S. derby in this epidemic. It was isolated from the asymptomatic individual, from the patient with relatively mild disease with a few watery stools, and from the patient severely ill with marked diarrhea, high fever, nausea, and vomiting with resultant aberations in fluid and electrolyte balance. In some, it may have been a secondary cause of the patient's demise. However, it must be pointed out that the majority of the individuals affected by this organism were asymptomatic or had mild symptoms. The patient with severe disease, for the most part, was suffering primarily from a chronic debilitating disease such as a malignancy or chronic gastrointestinal disorder. The following are brief records of the clinical features of four typical cases.

1. M. P., a 25-year-old male intern, was found to have a positive culture for S. derby on routine survey. This physician had been caring for a number of patients who had gastroenteritis due to S. derby. He had been asymptomatic and did not develop any symptoms during the 4 weeks that he harbored the organism and was off duty. There was no history of antecedent gastrointestinal disease.

2. W. W., a 48-year-old female, was admitted to the hospital because of suspected biliary colic. A complete evaluation of her gastrointestinal tract revealed only the presence of calculous cholecystitis. The patient had a negative stool culture for salmonella at the time of admission. Seventy-two hours following admission and after a cholecystogram, but before a complete gastrointestinal series was performed, she developed an episode of diarrhea with 8 to 10 loose, watery stools over a 36-hour period. Her stool culture was positive for S. derby. It was necessary to delay her surgery for approximately 6 weeks until her stools were negative for salmonella.

3. J. M., an 18-year-old student nurse, had an onset of diarrhea and abdominal cramps while working on a floor which had housed a number of patients with diarrhea. She was hospitalized and continued to have abdominal cramps and diarrhea consisting of 4 to 6 liquid bowel movements the first day and 1 or 2 the second day. Following this episode, she was asymptomatic. Her stool culture was positive for S. derby during the symptomatic period and remained positive for 3 weeks. She was restricted from patient care for 6 weeks, after which time she returned to her clinical duties. She was well until October 7, 1963, when she again developed gastrointestinal symptoms and again had a positive stool culture. Her symptoms during this episode lasted 36 hours, and her stool remained positive for 3 weeks. She was removed for the second time from patient contact. It is impossible to state whether this was a re-infection or an exerbation of salmonellosis.

4. J. A., a 62-year-old male, was first admitted to the hospital in October 1963 for an evaluation of anemia and back pain. A proved diagnosis of multiple myeloma was made, and he was treated with anti-cancer agents with some relief from his symptoms. His stools were negative for salmonella at that time. The patient subsequently had a recurrence of his symptoms and was again treated with cancer chemotherapeutic agents which are believed to suppress immunologic response.

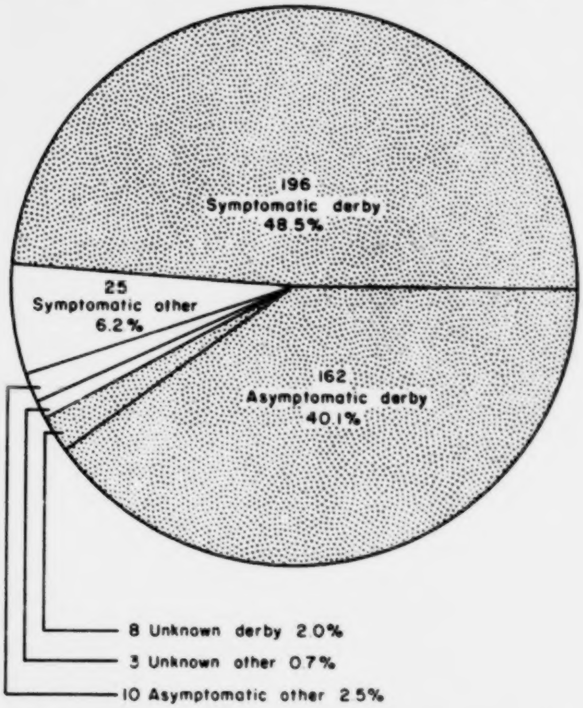
Six days after the institution of anti-cancer therapy he had a sudden onset of explosive diarrhea with rise in temperature to 104° and a fall in blood pressure to 90/50. Three blood cultures were made and were subsequently positive for S. derby. The patient was treated with antibiotics, recovered from his acute illness and was discharged from the hospital only to die suddenly at home 10 days later.

COURSE OF THE OUTBREAK IN PATIENTS

The first patient with S. derby enteritis had the onset of disease on March 6, 1963. Patients with diarrheal disease then appeared with increasing frequency until early April when it was obvious that a number of individuals were contracting diarrhea after admission to the hospital.

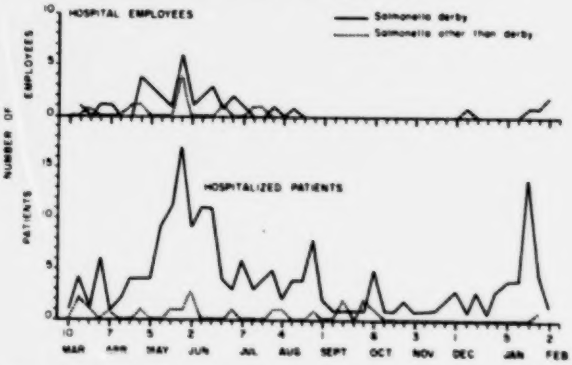
A total of 404 patients had positive fecal cultures for salmonella during the 12-month period from March 1963, through February 1964; 366 of the 404 were positive for S. derby. One hundred ninety-six (53.6%) of the total S. derby infections had symptomatic disease; whereas 162 (44.3%) did not have symptoms. The details concerning eight patients are unknown. The asymptomatic patients were cultured, for the most part, during the mass hospital surveys of April and May (Figure 2).

Figure 2.–*Distribution of salmonella positive patients*



The number of patients with diarrhea presumably due to S. derby increased slowly through March and April with a maximum number observed in May. The peak of the epidemic occurred during the week of May 26, 1963 (Figure 3). The outbreak reached a plateau for 3 weeks and then dropped to low levels during the summer. Approximately one case per week was registered during the fall months; but with the onset of winter, the number of cases rose again. The cause of these variations is not understood clearly but probably is due in great measure to variability in the emphasis and observation of strict hygienic measures throughout the hospital since the recognition of the outbreak. The factors causing the rise during the winter is understood less but may be due to some relaxation of hygienic practices and/or the continued presence of a reservoir of asymptomatic carriers among hospital employees.

Figure 3.–*Distribution of salmonella enteritis in hospital March 1963-February 1964*



As noted above, many patients with diarrhea due to S. derby had some underlying primary gastrointestinal disease. Some of these patients became ill following gastrointestinal surgery. Analysis reveals that 110 symptomatic patients had gastrointestinal disease alone or in combination with some other disease. Thirty-four patients had malignancy alone and 52 had other diagnoses. However, only 30 of the asymptomatic individuals had gastrointestinal disease, 27 had malignancy, and 105 had some other problem. The diagnoses of 8 patients were unknown. This marked increase in symptoms in patients with primary gastrointestinal disease is statistically significant (Table 3).

Table 3.–*Admitting diagnoses in patients from whom* Salmonella derby *was isolated*

Diagnosis	Symptomatic *S. derby* infection	Asymptomatic *S. derby* infection	Total
Gastrointestinal	72	26	98
Gastrointestinal and other	24	0	24
Gastrointestinal and malignancy.	14	4	18
Malignancy	34	27	61
Other.	52	105	157
Total diagnosed.	196	162	358
Unknown.	8	0	8
Total.	204	162	366

The majority of patients with S. derby disease have had mild symptoms. These were graded arbitrarily on the basis of +1 to +4 depending upon the severity of symptoms. Table 4 reveals that the great majority had mild symptoms consisting of diarrhea and abdominal pain; whereas only five had severe symptoms with impending death. These five also had underlying chronic disease which in itself was potentially fatal.

Thirty-four cultures of other body areas of fluids in addition to fecal cultures were positive in 27 of the patients. These included 3 wound cultures, 3 blood cultures, 5 throat cultures, 9 tracheobronchial tree cultures, 2 bile cultures, 2 intestinal fistula cultures, 9 urine cultures, and 1 cervical culture. The urine and cervical cultures, of course, could have been contaminated by the feces. The

Table 4.–*Severity of symptoms*–Salmonella derby *patients*

Symptomatic	Number of patients
+1 (diarrhea and abdominal pain)	131
+2 (diarrhea and abdominal pain and fever)	45
+3 (diarrhea, abdominal pain, fever, nausea, vomiting and dehydration).	15
+4 (+3 and shock or death)	5
Total symptomatic patients	196

majority of these 27 patients were moderately to severely ill and again had some underlying chronic debilitating disease.

There were 32 deaths among 366 patients. Nineteen of the patients had an underlying malignancy, 3 patients had severe gastrointestinal disease, and 10 had some other underlying disease. However, all of the patients who died had a chronic disease which in itself was life-threatening. No death occurred which could primarily be attributed to S. derby disease.

The number of salmonella species other than S. derby recovered from patients during this epidemic remained low but was greater than normally would be expected for an 11-month period. A total of 38 patients were positive for other salmonella, 25 (65.8%) were symptomatic, and 10 (26.5%) were asymptomatic, and in 3 the symptomatology was unknown (Figure 2).

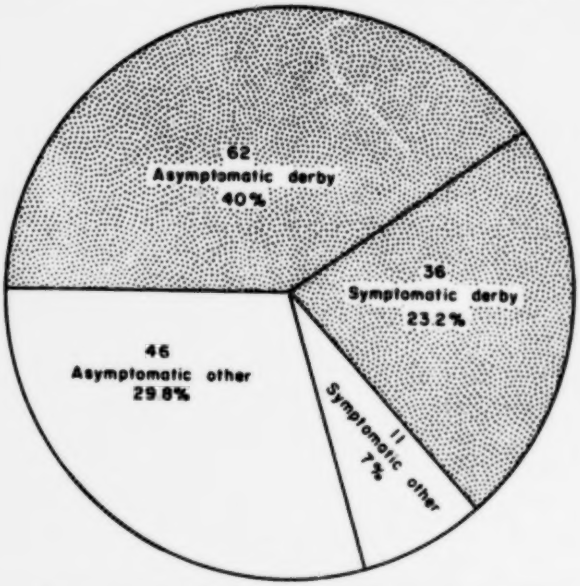
COURSE OF THE OUTBREAK IN EMPLOYEES

The first recognized employee with S. derby enteritis became ill during the middle of March 1963. This person, a student nurse, had, in November 1962, a previous episode of enteritis due to S. typhi-murium. Two other individuals then developed symptomatic disease in early April when the number of patients with S. derby was increasing rapidly. Cultural surveys of the employee group were instituted in late April and May. During this period, 12 employees had symptomatic disease; whereas 47 had positive stool cultures but were asymptomatic (Figure 3).

During this time, strict diarrhea surveillance was instituted and emphasis was placed on hygienic measures. All symptomatic employees and all people entering critical areas such as pediatrics, obstetrics, and the dietary department were cultured routinely. A few symptomatic and asymptomatic employees were noted each week until early June when the number decreased precipitously. This condition prevailed until late January 1964, when a cluster of symptomatic and asymptomatic employees were noted. This was coincident with the previously mentioned increase in the number of symptomatic patients.

The total number of employees found positive for all salmonella was 155. Ninety-eight employees were positive for S. derby, of which 36 (36.7%) were symptomatic and 62 (63.3%) were asymptomatic. Fifty-seven individuals were positive for other salmonella including 11 (19.3%) who were symptomatic, and 46 (80.7%) who were asymptomatic (Figure 4).

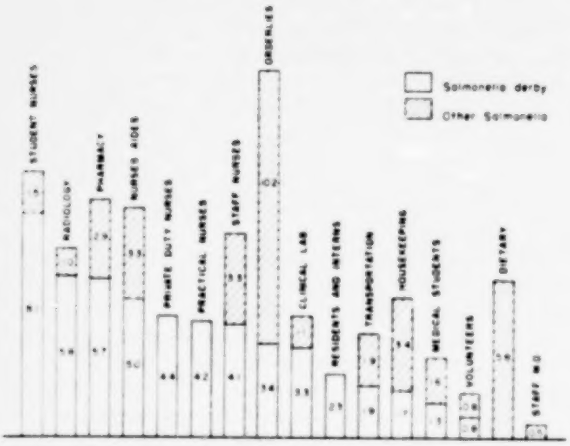
Figure 4.–*Distribution of salmonella positive employees*



The employee attack rate for all salmonella organisms is noted in Figure 5. Both symptomatic and asymptomatic individuals are included. Early in the outbreak, there was a suggestion that the closer the contact an employee had with patients, the higher the risk of contamination with S. derby was. Analysis of the data reveals that nursing personnel in general had an increased risk of contamination, with the risk of the student nurse group being the highest. It is to be noted, however, that almost all employees, except the pharmacy and clerical groups, contaminated by S. derby had close patient contact. Pharmacy workers pick up, clean, and in some cases, destroy items from the nursing stations which have been in intimate contact with patients, and therefore this group has an excellent opportunity for contamination. Four clerks in widely scattered areas had stools positive for S. derby, but an accurate attack rate could not be calculated.

The number of isolates of salmonella species other than S. derby also increased precipitously during the periods of mass survey in late April and May, but they remained relatively normal for the other periods of the epidemic. Two small intrahospital epidemics were noted during this period. A small number of salmonella group E positive employees was noted in late April during the first hospital survey, and a larger number with salmonella group C_2 was

Figure 5.–*Percent of salmonella attack rate, hospital employees, March-May 1963*



noted during the second survey in May. The personnel in both groups ate a large number of meals in the hospital commissary, and all those positive for salmonella group C_2 in the second survey ate roast turkey during the weekend prior to their positive culture. Figure 6 demonstrates the percentage distribution of all salmonella species isolated from employees during the two survey periods. Salmonella derby was the major serotype in both groups. However, the change in the other serological groups is interesting, particularly the increase of salmonella group C_2 organisms from 13 percent in April to 25 percent in May. One-quarter of the individuals comprising the salmonella group C_2 in May were from the dietary department. This was the first time in four complete surveys of this department that a large number of commissary workers had stools positive for salmonella. A small number of patients in the hospital also were noted to harbor salmonella group C_2 organisms in their gastrointestinal tracts at the same time.

Following the recognition of this outbreak, a number of raw turkey samples from the commissary were cultured over a 2-month period. The hospital obtained poultry from two purveyors at this time, and the turkeys cultured from one source were consistently positive for group B and C_1 salmonella (Table 5). The epidemiologic evidence is highly suggestive but not conclusive that these outbreaks originated from poultry in the dietary department. However, the evidence also postulates that S. derby

Figure 6.–*Percentage distribution of all salmonella in employee surveys*

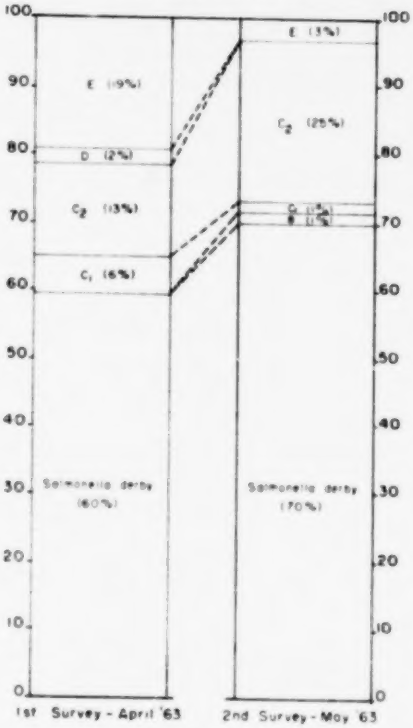


Table 5.–*Environmental cultures positive for salmonella*

[Total number of environmental cultures – 4,206]

Date	Type of sample	Location	Results
1963			
May 7	Surface swab	Water closet in student nurses' residence	*derby*.
June 8	Surface swab	Toothbrush holder in medicine chest in isolation room 18	*derby*.
June 8-Aug. 15	Surface swab	Uncooked turkey from dietary department	Groups B and C (not *derby*).
June 13[1]	Air sample–settling plate	Isolation room 21	*derby*.
June 13[1]	Air sample–settling plate	Isolation room 28	*derby*.
June 13[1]	Air sample	Isolation room 30	*derby*.
July 22-29[1] . .	Surface swab	Annex room 1255–water closet and wash basin	*derby*.
July 22-29[1] . .	Surface swab	Annex room 1256–water closet and wash basin	*derby*.
July 22-29[1] . .	Surface swab	Annex room 1260–wash basin	*derby*.
July 22-29[1] . .	Air sample.	Annex room 1260–settling plate	*derby*.
July 22-29[1] . .	Air sample–settling plate	12th Annex corridor	*derby*.

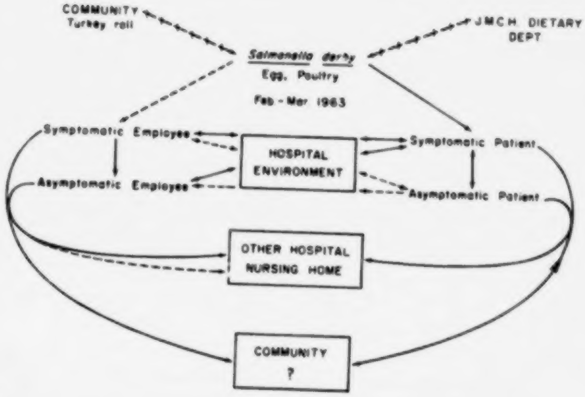
[1] These environmental samples obtained by CDC Technology Branch, Savannah, Ga.

was not introduced into the hospital via a food such as turkey in light of the fact that no dietary employee was ever positive for S. derby. Rather, the data speculates that the organism may have gained entrance by contamination of raw or undercooked eggs. The initial infection appears to have spread primarily to certain groups of patients and not to employees.

METHOD OF ORGANISM SPREAD IN THE HOSPITAL ENVIRONMENT

During the early epidemic period, several cases of recognized, acute, community-acquired S. derby infections were confirmed (Figure 7). These were due to contamination of turkey roll, and it was thought that the problem in the hospitals might be a reflection of a community problem. A study, therefore, was conducted on 639 consecutive private admissions in early June. The first hundred or so patients were cultured leisurely, but the last 500 were cultured in 1 to 6 hours after admission.

Figure 7.–*Epidemiologic pattern of* Salmonella derby *in hospital and community*



Ten of these patients harbored demonstrable salmonella in their stools. Seven of these organisms were S. derby, and three were of other serotypes. Four of the seven patients positive for S. derby had initial cultures taken at least 18 hours after admission and, therefore, could have acquired the organism following hospitalization. Three patients had positive stool cultures within 6 hours, but two of these had a prior recent admission during which time it is presumed they could have acquired the organism. One patient had no known contact with the hospital and appeared to have been positive for S. derby on admission. Three hundred ninety-seven of the above patients had repeat stool cultures within 96 to 120 hours after admission. Sixty-four (16.1%) of these patients had positive fecal cultures for S. derby. These statistics conclusively demonstrate that at the time of this study a significant percentage of patients were acquiring their organisms after admission to the institution. It also demonstrates that the community probably was not heavily contaminated with the organism at this time.

Attention then was directed to the mechanism(s) of organism dissemination when it became evident that intrahospital spread was occurring. Many possible sources of contamination and many fomites were cultured at various times throughout the first 6 months of the epidemic. These included food, water, ice, garbage, bedpans, bedpan sterilizers, water closets, sinks, tabletops, kitchen utensils, floors, ceilings, air vents, laundry chutes, carriages, and wheelchairs.

The total number of environmental cultures made was 4,206 (Table 5). Positive cultures were obtained from many areas within and outside the isolation units. Usually, however, an area found positive had recently been inhabited by a symptomatic, positive patient. This was true in the case of Room 1255 and Room 1256 which have a common interconnecting bathroom facility. The positive culture in Room 1260 is not explained as easily, but the positive corridor culture was taken in close proximity to Room 1255 and probably can be explained on the basis of air flow. The positive culture within the nurses' home is explained easily when the attack rate of the student nurse population is considered.

One source of contamination considered early in the epidemic was the inadequate sterilization of bed pans. A cultural survey of 384 bedpans from patients revealed that S. derby could be isolated from 8 of them. In 6, both the bedpan and the patient were positive, while in 2, the patient was negative. There was one occasion when the bedpan was positive, and the patient negative, but the roommate was positive. In 36 instances, the bedpan was negative, and the patient positive. These data suggest that positive patients significantly contaminated bedpans with S. derby. Additionally, it is suggested that a patient could, upon admission, receive a previously contaminated bedpan or his own pan may not have been returned to him after emptying. This situation was rapidly corrected and the bedpans sterilized after use.

Another source of contamination was the throats of the patients and employees from whom S. derby had been cultured in the feces. A total of 99 throat swabs were taken from patients or employees harboring salmonella: 85, S. derby and 14, other salmonella. Five throat cultures were positive for salmonella; four were S. derby and one, a group D salmonella. The hands of the employees, particularly those involved with patient care or food service, also were considered as vectors. Eighty-five pairs of hands from physicians, nurses, housekeeping, and dietary personnel were chosen at random on the isolation floors and cultured. None were positive. However, handwashing with soap, water, and iodofor dips was at its highest peak during this period and may have reduced the chance of a positive culture.

These statistics indicate that much of the hospital environment was heavily contaminated with S. derby from the middle of May until late July. They also demonstrate that other salmonella were introduced via the dietary department, but, for reasons unknown, did not disseminate as did S. derby.

Epidemiologic patterns suggest that the susceptible patient in the hospital is the individual with gastrointestinal or malignant disease. This patient readily develops symptomatic disease with varying degrees of diarrhea and sheds a large number of organisms into his environment. These subsequently contaminate nursing personnel, other patients, and fomites. The chance of contaminated individuals developing symptoms appears to be enhanced by at least two factors: first, the size of the organism inoculum, and/or second, the presence of primary gastrointestinal or malignant disease.

A contaminated but asymptomatic patient or employee may also spread the organism to other susceptible patients who, because of their subsequent diarrhea, then add a strong new link to the chain. The symptomatic employees also add to the contamination if they are admitted to the hospital or particularly if they are not immediately removed from patient contact.

Analysis of our data reveals that the initial cases were confined to the private service in one building (Thompson) (Figure 1). The infection then spread to an interconnected private service building (Foederer) and finally spread to the general ward service in the Old Building. The last two places to have recognizable contamination with S. derby were the pediatric ward service and the nursery-obstetrical service. This method of spread is the reverse of what is expected, and the reason for this reversal is the subject of further study. The data suggests, however, that the organism was carried from place to place by personnel and/or fomites. Finally, the entire institution was heavily seeded with the organism; and positive, symptomatic patients were observed in every nursing unit with the exception of the Nursery Service where positive but asymptomatic infants were observed.

Patients were discharged from the hospital with positive stool cultures, but care was taken not to send these patients to another hospital or nursing home. However, at least one incident is known of a previously positive patient discharged from one hospital to a convalescent hospital after 3 negative stool cultures. The patient subsequently developed diarrhea and was positive for S. derby; and the organism was noted to spread and cause symptoms in at least 5 of 50 patients in the convalescent hospital.

Some previously positive patients were discharged from the hospital with negative stool cultures and subsequently returned to the hospital for further therapy. They were found to be negative for salmonella on re-admission; but shortly thereafter, cultures were again positive, and they may or may not have developed symptoms of gastroenteritis. These patients were usually those with advanced malignancy or a severe gastrointestinal disease. It is difficult to say whether this is a reinfection or merely the exacerbation of an infection from which the organism was not recovered on readmission culture.

The status of the community in relation to S. derby at the present time is unknown (March 1964). However, at least four of the initially involved hospitals in the Philadelphia area are known to have had culture positive, symptomatic patients since the first of 1964. Our institution continues to have symptomatic patients, and one can presume that the community at large is now seeded with S. derby.

METHOD OF CONTROL

Following the recognition of the epidemic situation, every effort was made to locate the primary source of infection. When this failed and secondary spread became obvious, the entire hospital was alerted to the necessity of improving standards of personal and general institutional hygiene. This particularly applied to handwashing and to the care of contaminated linen, fomites, etc. New sinks were installed, multiple hand dip basins were provided and many "reminder" signs were posted throughout the hospital. Raw or undercooked eggs were removed from the menu when these were implicated as a possible primary source of infection.

Two floors (60 beds) were turned into isolation units: one, specifically for isolating any patient with diarrhea until etiology was known, and the second, for patients with positive cultures for S. derby. If patients were positive, they were either discharged home or moved to this isolation floor. Employees with diarrhea were removed immediately from patient contact until the etiology of their diarrhea was known. If found positive for salmonella, they were excluded from patient contact until their stools were consistently negative for salmonella organisms. This resulted in a trememdous manpower and economic loss to the hospital. Approximately 2,250 workdays were lost by the physicians and nursing personnel alone during the epidemic. The student nurse group accounted for over half of this total.

Rooms which previously housed positive patients or areas from which salmonella was cultured were specially cleaned. These were fogged and scrubbed with iodofor solution and remained closed for 6 to 12 hours prior to being reopened for patient care. Even after these decontamination methods, we were able to culture S. derby from some rooms. One room in particular appeared to be a focus of infection, as three consecutive patients revealed positive cultures despite the fact that the room had been fogged between each patient.

Numerous other control measures were instituted throughout the hospital, such as limitation of visiting hours, closing of lounges and roof gardens, restriction of patients to their rooms, and prohibition of food from outside sources. Laundry chutes were closed and stringent control measures were instituted in the handling of laundry on the floors. Carafes were steam sterilized and paper cups were substituted for glasses.

Special classes were conducted for all personnel, particularly for supervisory and administrative persons. Health department teams were provided by the city to help with the many problems involved. — All of these steps seemed to have some effect on the course of the epidemic with a gradual decline occurring during the late summer and early fall. However, despite all measures, we have been unable to eradicate completely the organism from the environment.

COMMENT AND SUMMARY

An extensive hospital-acquired outbreak of S. derby infections involved over 450 people in one hospital. The epidemiologic data suggested that the organisms gained admission to the hospital via a contaminated food, found fertile ground in several patient groups, particularly those with gastrointestinal or malignant disease, and rapidly contaminated the personnel, other patients, and much of the hospital environment. Secondary spread of the organism in the institution probably occurred by direct person-to-person contact, fomite contamination, and aerial dissemination.

This epidemic closely resembles an outbreak due to S. typhi-murium described by Datta et al. (4). They noted the work of McCullough and Eisele (7, 8) who stated that the ingestion of fairly large doses of certain salmonella strains were necessary to produce clinical illness but that even small doses gave rise to a transient carrier state. These phenomena, theoretically, would account for the epidemiologic pattern of the outbreak herein described. One only can speculate as to the reasons why S. derby spread with such apparent ease and why the other serotypes such as the S. muenchen seen in March did not disseminate.

A singularly important question concerns the relationship of this organism to the total problem of hospital-acquired infection. It has been shown that only one-half of those individuals contaminated by this organism developed symptoms. The majority of the symptomatic people had only a mild diarrhea, some nausea, occasional vomiting, and/or abdominal cramps. No patient died of primary salmonella disease; but a few people, very ill with malignant disease, had their lifespans shortened by the effect of severe diarrhea and dehydration. Rubbo in describing a pediatric outbreak in 1948 due to S. derby came to much the same conclusion. He stated, "The healthy infant may suffer no more than a mild diarrhea and slight vomiting or even be symptomless; whereas the sick child may present a far more serious syndrome, not infrequently terminating in death" (1). This organism, therefore, at present, albeit capable of producing quite annoying symptoms, and/or death in the chronically ill, is of less clinical and pathological significance in our institution than is the staphylococcus or some of the Gram negative bacteria. These latter organisms, of course, are capable of and have produced severe morbidity and mortality.

Salmonella derby is classified in a genus of organisms, the mere isolation of which is governed by public health statutes in many communities. Because of the association with typhoid fever (S. typhi), any publicity concerning salmonellosis may alarm the public. The regulations governing individuals and institutions affected by salmonella serotypes are quite restrictive and rightly so, at the onset of an epidemic. However, when the clinical disease proves mild, these regulations may impede, to a degree, over-all evaluation and investigation.

However, the reported increased incidence of salmonella contamination and disease in the United States and the rest of the world makes the careful clinical surveillance of this entity mandatory, particularly in relation to the chronically ill and the very young (9). Surveillance may be decreased or even disregarded when its effect is unnecessarily severe and penalizes a person, hospital, or an agency. We should re-evaluate the total problem and, in particular, each individual outbreak from a combined bacteriologic, pathologic, clinical, and epidemiologic viewpoint, in order to allow better understanding of the many unknowns, in the salmonella carrier state as well as the disease, as they occur in hospital-acquired epidemics.

ACKNOWLEDGMENT

The authors are indebted to all those who individually and collectively comprise the Jefferson Medical College and Medical Center for their help and understanding and, in particular, to Miss Mabel C. Prevost, Assistant Director, Jefferson Medical College Hospital. We also acknowledge the help of the United States Public Health Service, Salmonella Surveillance Unit, Communicable Disease Center, Atlanta, Georgia, particularly Dr. Eli Friedman and Dr. W. Eugene Sanders, and we express our appreciation to Dr. Kristine K. Knisely, Department of Public Health, Philadelphia, Pennsylvania.

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Organization of Laboratory in an Epidemic Situation

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Frank J. Sweeney, Jr., M.D.[2]

Prior to the epidemic situation at the Jefferson Medical College Hospital, approximately 150 fecal specimens were processed monthly in the hospital bacteriology laboratory. These specimens were cultured for salmonella and shigella on MacConkey agar, salmonella-shigella agar, and in selenite F broth. The number of isolates of salmonella species over the past several years has averaged two per month. After isolation, the organisms were typed seriologically with grouping sera and sent to the Commonwealth of Pennsylvania Laboratory for species identification.

During the month of March 1963, it was realized that a greater than average number of salmonella isolates were recovered (16), and practically all of these were group B salmonella. As we became aware of the existence of a potential epidemic problem within the hospital, various possible sources of infection were cultured.

During the second week in April, all dietary personnel were cultured in order to rule out this commonly implicated source of salmonella contamination. In addition, a large number of environmental samples were examined; most of these were aliquots of prepared and unprepared foods, but some were swabs taken from food preparation areas. At this time, cultures pertaining to the salmonella survey were separated from the routine cultures for ease in numbering and reporting. The specimens, consisting of a stool and a rectal swab from each dietary employee, were cultured as described above. The dietary personnel again submitted rectal swabs and fecal specimens for culture during late April, and an increased number of other hospital employees and patients were cultured throughout April. During this month, a total of 890 cultures were processed. Sixteen new isolates of Salmonella derby and four isolates of other salmonella species were reported. None of the S. derby isolates was recovered from dietary personnel.

During the first week of May, a decision was made to conduct a survey of a large part of the hospital population. It was realized that additional laboratory assistance would be needed to cope with the large numbers of cultures anticipated. Upon request, a professional microbiologist and an experienced technician were sent from the Veterinary Public Health Laboratory, Communicable Disease Center, Atlanta, Georgia. At their recommendation, certain basic changes were made in the methods employed to culture fecal and environmental specimens.

A single swab was obtained from the patients or personnel and immediately placed in tetrathionate broth. After incubation at 37°C, the broths were subcultured to brilliant green agar plates for salmonella isolation. Using this method, an approximate 48-hour period is required before the results of the culture are known. At times during the epidemic, especially during the first large survey, the broths were subcultured a second time after prolonged incubation. Incubation periods longer than 24 hours result in an increase in the number of positive cultures. However, the danger of cross-contamination of tubes is always present especially when unsupervised or inexperienced personnel are subculturing.

After the arrival of the Communicable Disease Center personnel, a specific identification of S. derby could be made serologically within a few hours after the organisms were isolated. We were able to separate quickly the S. derby strains from other group B organisms.

Within an 8-day period, from a total of 2,319 cultures, 82 new isolates of salmonella were recovered from patients and personnel. One environmental sample was reported positive from the student nurse residence. All of the laboratory procedures were performed in the hospital bacteriological laboratory without interruption of the regular services offered. However, this necessitated utilizing the full time and overtime services of 5 bacteriology technicians plus overtime services of 13 other technical personnel.

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During the next two weeks, medical student surveys were conducted and yielded 343 cultures. Five students were found to harbor salmonella; cultures from two of the five were positive for S. derby.

On May 23, the Philadelphia public health officials requested that all hospital personnel be resurveyed. Accordingly, assistance again was requested from the Communicable Disease Center, and three people arrived to assist with this overwhelming task.

At this time, it was agreed that it would be best to remove the processing of the specimens from the routine laboratory to the recently established Salmonella Surveillance Unit which was situated on the same floor but in the adjacent interconnecting building. In 3 days, a laboratory was established to which all specimens pertinent to the epidemic were directed. All the specimens obtained from personnel during the next several weeks were taken to the Salmonella Surveillance Unit; and, therefore, the proximity of the laboratory to this unit was of prime importance. Prior to the establishment of the unit, multiple areas of the hospital complex were used for culturing, and the specimens were transported to the laboratory.

Old unused tables and desks were brought from storage and were utilized for the laboratory work areas. Incubator space proved to be a difficult problem. One large incubator was moved from the routine laboratory, and two old incubators were brought from storage and returned to service. Another incubator was obtained on loan from a generous local scientific equipment distributor. The overflow racks of tubes were incubated in the routine laboratory.

Illuminating gas outlets were not available in the room utilized as a laboratory, so large refillable containers of butane were acquired from which four to five Bunsen burners could be fed at one time. Culture tubes, culture tube racks, reading lights, water baths, pipettes, Bunsen burners, and a variety of equipment were borrowed from various laboratories in the Jefferson Medical College. Two carriers for transporting materials to and from the routine laboratory were secured.

The procurement of an adequate supply of dehydrated culture media provided another problem. When local distributors could not supply our needs, several manufacturers were contacted directly, and they rapidly supplied the materials.

The suspected epidemic of S. derby gastroenteritis in multiple Philadelphia hospitals prompted a request from the local health officials that cultures from other institutions be processed in the special laboratory at the Jefferson Medical College Hospital. This provided all institutions with expert bacteriological evaluation in a central area. It precluded the necessity of interhospital movement by bacteriologists from the Communicable Disease Center. Technical assistance was provided by the cooperating hospitals.

The greatest deterrent in processing large numbers of cultures lies in media preparation. Because cultures from other hospitals were being processed at Jefferson, a large amount of tetrathionate broth and brilliant green agar plates was prepared by the laboratories of the Philadelphia Department of Public Health. This proved to be quite important psychologically, because it removed one burden from already overworked personnel.

In order to culture expediently large groups of specimens, numbering became a serious problem. Different series were coded by letters and were not allowed to exceed 1,000 specimens per series, because it was found that many errors occurred when more than three digits were employeed. Certain confusing letters of the alphabet were avoided, and easily written letters of the Greek alphabet were used.

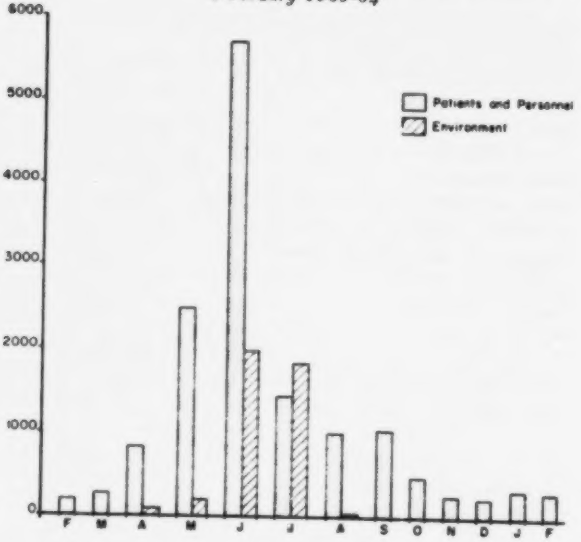
Record keeping became the most difficult procedure. Reporting salmonella-positive patients and personnel was accomplished easily and rapidly. However, the daily following of cultures from employees and patients who previously had been reported positive proved extremely difficult. When several different series of cultures were taken simultaneously, it was almost impossible to know in which series a particular individual's report could be found. Various colored laboratory slips were used to facilitate the finding of these reports. This was particularly essential in the case of employees who had been removed from duty until a certain number of negative reports were obtained. For example, the regular bacteriology form is green; and, therefore, a brown laboratory form was employeed when the specimen came from a known positive employee. Later on during the epidemic other colored laboratory forms were used to signify certain studies.

Tubes of tetrathionate broth were prenumbered before they were placed at the culturing stations. The same numbers were placed on the laboratory form at the time the specimen was obtained. After a rack was filled, it was taken to a central area, where the date and time were electrically stamped on the laboratory form. The person stamping the form made certain the forms and tubes were in numerical order and then removed a carbon copy of the form for a central file. The tubes and the remaining two copies of the laboratory forms then were taken to the laboratory.

Ringed binders were used in the laboratory to house the work sheets employed for organism identification. The request forms were filed numerically in boxes until they were completed. Positive cultures were reported first and given as soon as possible to the project coordinator. Negative cultures were reported at a later date when all questionable positive isolates had been further identified. The completed original and carbon copies of all request forms were taken to the project coordinator. At a later date, the carbon copy was returned to the laboratory in order that the individual's name and pertient information could be placed on the work sheet with the result of the culture.

The second survey covered the period from May 24 through June 30. A total of 7,357 specimens from Jefferson Medical College Hospital were cultured, and 2,706 were processed from the other Philadelphia hospitals. A total of 241 new salmonella isolates were recovered from employees and patients at Jefferson during this survey. Fifteen cultures positive for S. derby were reported from a variety of environmental samples, and six isolates of S. typhi-murium were recovered from raw turkeys which had been cultured in the commissary (Figure 1).

Figure 1.–*Total numbers of specimens processed February 1963-64*

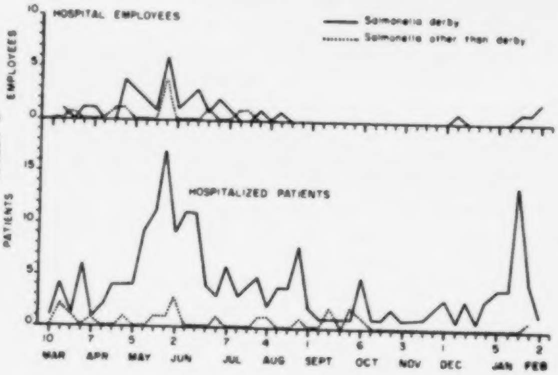


During the second week of June, Communicable Disease Center personnel from Savannah, Georgia, collected environmental samples by means of impression plates, settling plates, and an air sampling machine. A large number of environmental samples were taken from the isolation areas. Four of the 15 environmental samples reported positive for S. derby were recovered from settling plates and from samples obtained with the air sampling machine. The same personnel returned to Jefferson during the third week of July to collect samples from high incidence areas other than the isolation floor. Eight isolates of S. derby were recovered at this time from settling and impression plates.

For the most part, only symptomatic patients and personnel were cultured after early July. The main exceptions were maternity patients who had admission and discharge swabs taken in July, August, and September. Student nurses and medical students were screened throughout the epidemic before going to maternity and pediatric services.

The numbers of specimens submitted to the laboratory totaled 1,015 during the month of August and 1,018 during September. The Salmonella Surveillance Unit was closed and the emergency laboratory disbanded on October 12, 1963; and subsequently all fecal specimens were cultured in the routine laboratory. After October, the number of specimens for salmonella isolation decreased precipitously compared with the previous 6 months. The number of new isolates of S. derby remained low during October and November. However, a significant increase in the numbers of symptomatic patients and personnel was observed at the end of December and continues through the present time (March 1964) (Figure 2).

Figure 2.–*Distribution of* Salmonella enteritis *in hospital March 1963 – February 1964*



From July 1963 through February 1964, 244 new isolates of salmonella were recovered from a total of 7,038 cultures from patients, employees, and environmental samples processed.

Under normal conditions, approximately 1,800 fecal specimens are cultured during a 1-year period in the laboratory at the Jefferson Medical College Hospital. However, during the year, from March 1, 1963, through February 29, 1964, a total of 21,202 fecal and environmental samples were processed.

A total of 630 primary isolations of salmonella from patients, employees, and environmental samples from Jefferson Hospital were reported during this 1-year period (Figure 3). Many patients and employees had numerous repeat positive and/or negative cultures: e.g., one student nurse had 24 consecutive positive cultures, and another hospital employee had 54 consecutive positive cultures. The total number of salmonella isolates including repeat cultures has not been determined. The length of carrier time for S. derby in different individuals during the epidemic is the subject of further study.

Many times over the past months, we have reviewed the problems which we encountered during this epidemic. The following suggestions might aid in the investigation and control of future epidemics due to similar agents. See Figure 4.

A. Record Keeping:

   1. Acquire a well organized secretary for general over-all supervision of clerical personnel and particularly to oversee the numbering of cultures and the checking of lists.

Figure 3.—*Total numbers of new positive isolates recovered March 1963—February 1964*

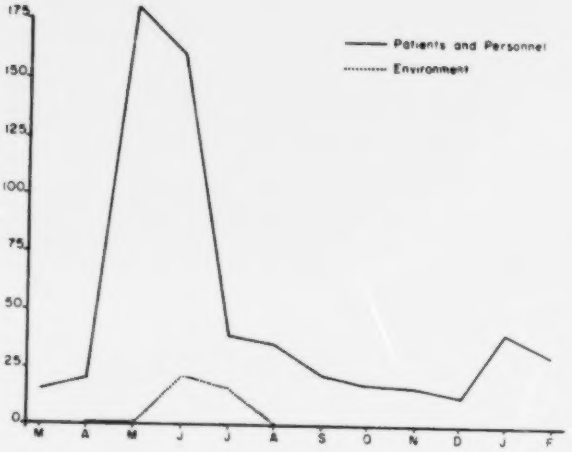
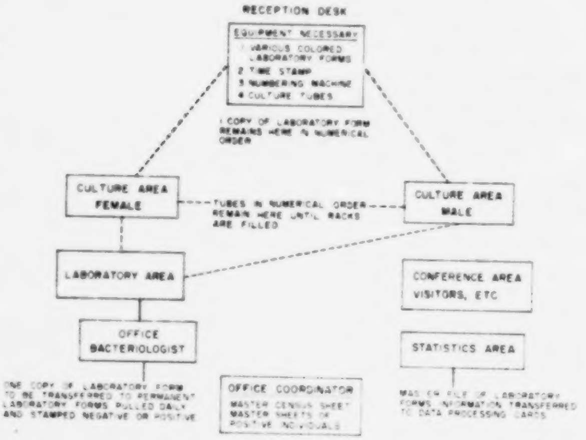


Figure 4.—*Diagram of Surveillance Unit*


RECEPTION DESK
EQUIPMENT NECESSARY
1 VARIOUS COLORED LABORATORY FORMS
2 TIME STAMP
3 NUMBERING MACHINE
4 CULTURE TUBES
1 COPY OF LABORATORY FORM REMAINS HERE IN NUMERICAL ORDER
CULTURE AREA FEMALE
TUBES IN NUMERICAL ORDER REMAIN HERE UNTIL RACKS ARE FILLED
CULTURE AREA MALE
LABORATORY AREA
CONFERENCE AREA VISITORS, ETC
OFFICE BACTERIOLOGIST
STATISTICS AREA
ONE COPY OF LABORATORY FORM TO BE TRANSFERRED TO PERMANENT LABORATORY FORMS PULLED DAILY AND STAMPED NEGATIVE OR POSITIVE
OFFICE COORDINATOR
MASTER CENSUS SHEET
MASTER SHEETS OF POSITIVE INDIVIDUALS
MASTER FILE OF LABORATORY FORMS INFORMATION TRANSFERRED TO DATA PROCESSING CARDS

2. Ascertain that no specimen is taken to the laboratory before the report sheet is stamped for date and time of receipt and a copy is pulled.
3. Record each departmental, patient, or environmental survey on a different colored slip to facilitate handling.
4. Pull laboratory forms daily, indicate positive reports, and give to coordinator.
5. Take laboratory forms accumulated each day to laboratory and record pertinent information in the permanent laboratory record.
6. Total each group survey for percent of participating personnel and calculate attack rate.
7. Keep master sheets on a daily basis for all positive patients and personnel.
8. Fill out a data processing card for each positive patient or personnel, and keep a record of all subsequent culture reports whether they be negative or positive.
9. Have a physician record any pertinent information on the data processing cards on all new patients or employees on a daily basis.
10. Maintain a daily census sheet of the isolation area.

B. Personnel required for such an epidemic include:
   1. Physician-coordinator who is responsible for the isolation of patients, institution of control measures, epidemiologic surveys, and general supervision.
   2. Professional microbiologist who would establish a laboratory if existing facilities are not adequate, oversee laboratory procedures, and assist the coordinator in epidemiological surveys.
   3. Bacteriology technicians well trained in organism isolation techniques and serological procedures.
   4. Supervisory secretary.
   5. Clerks
      a. For checking personnel lists and assigning laboratory numbers.
      b. For numbering tubes of media and processing forms of incoming specimens.
      c. For signing out laboratory forms and recording pertinent information in the laboratory books.
      d. For preparing master sheets and totaling and computing percent of participation and attack rate for each departmental and patient group.
   6. Nurses and physicians for culturing personnel.
   7. Secretary for telephone work, typing reports, and other general secretarial duties.
   8. Resident physicians for accumulating information concerning each patient and employee and making entries on data processing cards. Same residents may be used for obtaining specimens for serological studies, throat cultures, and admission cultures on patients.

C. The following materials are needed:
   1. Stamp sets
   2. Electric date and time stamping machine
   3. Reduplicating number stamper
   4. Triplicate laboratory forms in 10 to 15 different colors
   5. Boxes for sorting laboratory forms
   6. Numerous pencils and pens of different colors
   7. Typewriter
   8. IBM or departmental sheets of all personnel

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Panel Discussion

Dr. Philip S. Brachman, Chairman,
Dr. Edward W. Hook, Dr. Kenneth W. Newell,
Dr. W. Eugene Sanders, Jr., Dr. Frank J. Sweeney,
and Dr. Robert I. Wise

DR. BRACHMAN: We have a number of interesting questions. We would like at least for the first part of this session to discuss the hospital aspects of salmonellosis.

> QUESTION: How long do you keep a patient hospitalized who is still excreting salmonella organisms but is free of symptoms, both of his original illness and salmonellosis?

DR. HOOK: We do not make any effort to keep patients in our hospital until they cease shedding the organisms. Considering Salmonella derby infections, this would be an extraordinary thing to do, since such a large proportion of the patients continue to excrete organisms for so long a time. If their general medical condition no longer requires hospitalization, they are discharged from the hospital.

> QUESTION: If a patient who is still shedding organisms in spite of antibiotics is going back to a large household, what instructions do you give him?

DR. HOOK: First, about antibiotics, there is not a tremendous amount of evidence, in terms of controlled studies, concerning the effect of antibiotics on the course of salmonella gastroenteritis. I think the evidence that is available indicates quite clearly that regardless of the serotype involved, no antibiotic therapy really alters the clinical course of gastroenteritis caused by salmonella or alters the length of time during which organisms can be recovered from the intestinal tract. I am hedging a bit in terms of one drug, the new semi-synthetic penicillin, ampicillin. It has not been thoroughly evaluated in this regard, but I suspect the same will hold true for it.

As for instructions to the patient, they are the general measures that you would stress with any patient who might spread any type of infection to members of his family. For example, we emphasize the importance of handwashing and other usual hygienic measures. We recommend that the patient not be involved in the preparation of food.

DR. BRACHMAN: Do other members of the panel have additional comments to make concerning this subject?

DR. SWEENEY: This is essentially what we did during the course of the S. derby epidemic at our hospital. It would have been impossible for us to keep the patient with salmonellosis in the hospital until he or she was free of the organism. We attempted to send the patients home and to instruct them and their families in the hygienic measures directed to prevent the spread of salmonellae. However, we tried not to send these people to other institutions such as nursing homes, convalescent homes, or other hospitals. If they had to have further care, we kept them in the hospital until they were culturally negative or until we could arrange to send them to a relative's home.
Dr. Hook mentioned ampicillin. Our experience with this drug is not very wide, but we have had failures. This is particularly true in our student nurse group where we treated a few girls in an attempt to get them back to work.

DR. TAYLOR: I agree fully with everything that has been said. We have had a certain amount of experience with schoolage children being allowed to go back to school after recovery from symptomatic disease, even though they were carriers. Never have I had a recrudesence of symptoms from a schoolchild going back to school.

DR. NEWELL: I think that from what evidence we have, we must consider the possibilities that (1) the S. derby involved in the interstate outbreak may be a special strain, (2) it may be spreading in an unusual way in the affected hospitals, or (3) it can cause human infections with a smaller dose. I know it is very hard to make the distinction not just between S. derby and other salmonellae but possibly between this S. derby and other S. derby's that we are finding elsewhere in the community.

While these possibilities still exist, I think we have to assume that it is easy to become infected after a contact with someone with this particular strain. I think the same precaution should be advised as with someone who has a highly infectious respiratory illness.

DR. GLASSMAN: Would any member of the panel care to speculate on the dose-response relationship between the host, man, and the parasite S. derby? Such thoughts might assist in evaluating the role played by the various potential routes of transmission. For example, if billions of organisms are required for infection of man, I would question the importance of the airborne route in naturally acquired infection.

DR. COCKBURN: This whole question of dose response to salmonellae is one which needs a lot of reinvestigation. If I am not mistaken, S. derby was one of the strains used by McCullough and Eisele (1) in their studies; and, as I recall, they found that S. derby was a serotype for which an extremely large dose was required to cause sickness. What dose was required to cause infection I do not remember.

I see Dr. Newell's point of view that this S. derby strain seems to behave in rather a peculiar fashion in the hospital outbreak that has been described. I agree with Dr. Taylor that, in the United Kingdom, we never have had any experience of any spread of infection from someone who had no diarrhea but who was known to be infected and was sent back into his own environment. The only precaution we advised, as far as I remember, was that they should not go back into an environment in which they were preparing food. To go back to school, or to work was all right. Because they were known excreters, we felt they were safer in some ways, than people about whose excretory situation we knew nothing.

DR. HOOK: I just wanted to raise a question. I think it may be a bit unfair to compare this particular strain of S. derby with another strain, for example, the one studied by McCullough and Eisele. The differences between this strain and that strain may be like the difference between this strain and a completely different salmonella strain. It seems to me that the outstanding thing about this particular epidemic is the communicability of the organism. I think it is quite possible that it might have been spread by food in many instances; but I think it is quite clear from the observations that have been reported, that something else is involved, some other method of spread.

One final point, and that is about the relationship between strains of a given salmonella serotype. If I am not mistaken, there are some people who feel that certain of the S. typhi-murium strains, phage type 27, behave differently from other phage types of S. typhi-murium. I believe Dr. E. S. Anderson made this point in connection with the outbreak at Hammersmith, which was associated with this type of prolonged spread, apparently by contact.

DR. BRACHMAN: I wonder if Dr. Cockburn and Dr. Hook might like to comment on whether the chronicity of this epidemic, as demonstrated at the Jefferson Hospital and in several other hospitals, could be related to dose response.

DR. COCKBURN: I do not think that this epidemic is very unusual. The S. derby outbreak which Rubbo (2) investigated in Australia went on for a very long time. The numbers of cases and excreters were large. It spread into the general community and persisted for a long time. McKerras and McKerras (3) reported another outbreak due to S. bovis-morbificans which behaved very much in the same way in a hospital population and spread into the community. Someone mentioned here that S. tennessee spread through a hospital over a 4-month period, resulting in many carriers, but no one developed clinical symptoms of gastroenteritis. (4)

In regard to the outbreak of S. typhi-murium, phage type 27, disease mentioned by Dr. Hook, there were approximately 150 cases and 160 excreters. Anderson stated he felt this particular phage type differs from others, but I see no evidence to support this. So I do not think there is anything unusual about the S. derby outbreak. I do not think it has gone on too long.

DR. TAYLOR: There is a point which I would like to mention, too, in support of Dr. Cockburn. The proportion of cases with symptoms to symptomless cases is much like what one would predict for a hospital outbreak with any of the salmonellae.

DR. HOOK: I am astounded at these comments. You are willing to accept the fact that this is person-to-person spread?

DR. TAYLOR: Yes.

DR. HOOK: Well, let me ask you, do you not consider this ususual?

DR. COCKBURN: No.

DR. HOOK: I think that certainly person-to-person spread has been recognized. I am aware of only three reported outbreaks involving adults in general hospitals where the disease was spread person-to-person and the outbreak continued for a considerable length of time. These are the one at Hammersmith, previously discussed; the one in Wisconsin (5), and the current S. derby outbreak. There may be others, but the reports of Rubbo and the one by McKerras concerned outbreaks which involved infants and were in nurseries. I think this is an entirely different situation.

DR. LASHOF: I want to ask specifically, when you were treating the patients with antibiotics, whether the stools were negative for salmonella just during actual treatment or for a more prolonged period of time; I know antibiotic therapy has not been effective in modifying the carrier state.

DR. HOOK: Sometimes the patients become negative during therapy and then may remain negative or become positive again when the therapy is discontinued. We have also had instances and others have been reported in which people continue to have positive stool cultures during chloramphenicol therapy. We have had a rather good experience with ampicillin in typhoid carriers. Among 23 patients followed very carefully while on this therapy, the stools from all of them became negative, at least so that we were not able to detect organisms even though we added penicillinase to the culture during the period of therapy with ampicillin. When ampicillin is discontinued, a large proportion of the patients then regain the organism in their stool.

DR. LASHOF: If you could keep the stools of patients negative for salmonella while they are on therapy, would this be a feasible measure we should consider using in the hospital to try to prevent hospital spread?

DR. HOOK: I think it is a very reasonable thought, though it would involve treating a tremendous number of people.

DR. SWEENEY: I do not agree with this last statement. The number of people who would have been treated with antibiotics during our outbreak would have been considerable. The cost would have been great, but more important the risk of toxicity from an antibiotic in a group of patients this large would have been high. I would anticipate some problems.

DR. SANDERS: I agree with Dr. Sweeney. I think there are two people in the audience who have had some experience that might help us discount the use of other antibiotics aside from chloramphenicol and ampicillin. I wonder if Dr. Wikingsson would comment about her experience.

DR. WIKINGSSON: We had asymptomatic patients at Inglis House, Philadelphia, and took a small group to whom we gave a course of treatment with tetracycline and another small group in whom we used sulfonamides. We cultured the patients daily, and found that some of them appeared to clear during the course of the tetracycline; but, very shortly thereafter, cultures were again positive. We could not be sure then whether or not it was a reinfection or had merely been a suppression for that period of time. We gave some a second course of tetracycline and pretty much the same thing happened again. Our results using sulfonamides (triple sulfa) were similar.

DR. SANDERS: Dr. Isenberg, would you care to comment?

DR. ISENBERG: It has been our experience at Long Island Jewish Hospital, New York City, that at least 50 percent of people who are convalescent or asymptomatic carriers actually have increased numbers of salmonellae in their stools after therapy is completed. This is regardless of whether they were treated with tetracycline, chloramphenicol, sulfonamides, or any other antibiotic. We have the impression which I think some people here may share, that especially during the carrier state and with nonsystemic salmonellosis, antimicrobial agents play a minor role, if any, in controlling the disease, or specifically, the number of salmonellae in stool. I do not think that in a hospital environment, especially where there is still the question of staphylococcal disease, it is a good idea to put people on antibiotic prophylaxis for salmonellosis.

QUESTION: Is it necessary or to be recommended that cases of salmonellosis in hospitals be kept in strict isolation, utilizing gowns, masks, and so forth? Does this really help beyond the routine hygiene and sanitation by hospital personnel?

DR. SWEENEY: I think that it is necessary to isolate patients or employees who are producing large quantities of stool. Whether or not this does any good, we are not sure, perhaps it makes us feel better.

The problem of the asymptomatic employee who has a positive stool culture for S. derby is somewhat different. We have allowed individual employees who are intelligent and capable of carrying out basic hygienic principles to return to work in certain areas. We, of course, have not permitted them to handle or prepare food and have restricted them from highly sensitive areas such as the nursery, obstetrical service, etc. We have not observed any problems when we have permitted this kind of activity.

QUESTION: How many successive negative stools should be required before assuming a patient is no longer a salmonella carrier?

DR. HOOK: I do not feel that there is adequate information in the literature or in our experience to say how many negative stool specimens are required to assure us that a patient is indeed negative. We have had some experience in New York City with typhoid carriers who have had negative stool cultures for eight or nine consecutive years. They have then turned up on the 10th year with a typhoid organism of the same bacteriophage type. So there is little evidence to say how many cultures indicate true negativity.

We have in progress several studies involving serial repetitive cultures of individuals, and we hope to get some meaningful data from these, but we still do not have enough cultures accumulated to permit valid analysis.

QUESTION: When can the food handler who sheds salmonella resume his occupation?

DR. TAYLOR: I think one has to tackle this problem in a practical way. You cannot withhold a man from earning his living forever, and in the United Kingdom, we demand three negative stool cultures taken at 3-day intervals.

DR. SLUTSKY: Just a comment with regard to food handlers. In the State of Illinois and in Chicago, food handlers are required to submit four stool specimens collected within 72 hours of each other. We have initiated a surveillance program on approximately 65 food handlers who are or were salmonella carriers and have found that close to 50 percent of this carrier group who have submitted the required four negatives, are again positive for salmonella 5 to 6 months later. We have checked their hospital charts and spoken to the attending physicians and have not found that the medications used have any advantages.

DR. SANDERS: We have completed one study following an outbreak of salmonellosis that occurred after a political rally in the Midwest, where there were some 313 individuals infected. (6) About three quarters of those were symptomatic. We went back 8 months following that outbreak and obtained one stool specimen from each patient and found 7 of 300 still carrying the same S. typhi-murium phage type organism. With S. derby infection in one institution, studied similarly, we found 33 percent of the patients carrying the organism 6 months after the suspected initial infection. I think we should say carriers do exist, at least up to 6 months with S. typhi-murium and with S. derby. What significance these carriers have as a public health problem once they are back in the community is another question.

DR. WISE: The dilemma that confronts us is one for which we need guidance and help. Everybody on this team has a responsibility. One that has concerned me has been the need of more knowledge. The only way we can obtain more knowledge, of course, is to investigate the problem that confronts us. But when we investigate the problem, we find a number of people who are asymptomatic. The asymptomatic carriers are removed from work and suffer economic hardship because of public health regulations.

In the stuvey of our own medical staff, one of the physicians had a positive culture for salmonella. It was not S. derby, but he was removed from patient contact. His occupation became difficult for him to pursue and inactivity became an economic loss to him. We have had emotional problems in students who have been removed from work and study in school. This has resulted in tremendous difficulty with their families.

The question that I have been thinking about is how do we work together to gather more information about epidemics and yet not harm the people involved in the carrier studies. If we seek to find more asymptomatic cases, then we have the problem of what to do with the ones we find. If we do not seek to find cases, there is safety in ignorance with our present public health regulations.

DR. TAYLOR: We were concerned with small babies who contracted salmonella infection in hospitals. The last thing I think one should ever do is to keep a healthy baby in an institution when it has a perfectly good family to go back to. So these babies were allowed to go back to their families with the families knowing that the babies were excreting the organism. We know from our experience that the babies who contract salmonella infection at an early age, say within the first month of life in the hospital, are likely to excrete that organism for many months; I usually say about 14 months. The few families involved were watched, and other members of the family picked up the organisms in turn. Sometimes the mother was positive, sometimes the father or some of the other children. But by and large none of the other members of the family had any symptoms.

One wonders, perhaps, in instances where the infection and risk are known and extra precautions are taken, whether one can exert enough control in surroundings where there are healthy individuals to stop disease from occurring in the other members of the family.

DR. SILLIKER: It just occurred to me with respect to this question of the food handler, that if it is accepted that he may be an excreter for a long period of time, maybe too much emphasis is being put on negative stools. Perhaps if the same effort were put into properly educating him in the proper handling of food, that the effort might be better spent.

DR. WINTER: In view of the recent S. derby incident, we are not so sure whether the classical hand-to-food-to-mouth transmission is the only method of transmission involved. I was quite interested in Dr. Sweeney's 5 percent positive throat cultures among the patients who were excreting the organism in their stools. I would like to make a plea to everyone who has the facilities and who deals with these outbreaks, to culture throats and noses and also to look for other potential routes of dissemination of the organism.

DR. HOOK: I just want to add one bit of information. We have not had an outbreak of S. derby infection in our hospitals, but we have had two or three patients admitted to the hospital who acquired the infection at other hospitals. One of the adult patients did have a positive throat culture, and we estimated the number of organisms in the patient's throat to be well over 10,000 organisms per 1.0 ml. of throat washing which was obtained with 10 ml. of diluent.

DR. COCKBURN: The point I would like to make concerns the isolation of salmonella from the respiratory tract. This is a very important point which does need more investigation. What bothers me about it is that in an environment in which there is a good deal of infection, the significance of isolating the organism from the respiratory tract is difficult to interpret. The organism may be in the dust. It may get onto the respiratory mucous and simply be lying there more less as a passenger on its way into the digestive tract. It is something we need to know more about before we start basing preventive measures on it.

DR. FOTER: I would like to point out that the problem of the return of food handlers to their work following infection involves much more than just human salmonella infections. This question has been raised in connection with the return of workers to the food handling line following hepatitis. The problem has also been posed to us by health authorities from the Philippines and Hawaii regarding the return of food handlers to work following amebic dysentery infection, and several years ago at a meeting sponsored by the Air Force on the Biologistics of Space Flight the question arose whether astronauts or pilots should be allowed to return to their duties if they were shedding organisms following an enteric infection.

My only point in citing these examples is that the problem is much broader than just salmonellosis in man. In time, we are going to have to come up with some public health guide lines for health authorities to follow; and, again, I would hope the CDC would give this problem close scrutiny and provide us with guide lines to follow, not only with salmonella but with other human enteric infections.

DR. SLUTSKY: Is there any way to explain why S. derby was restricted primarily to the Northeastern States and to the one other peculiar situation in the Midwest, namely Chicago?

DR. BRACHMAN: If we accept the vehicle of infection concept that has been discussed previously, this would explain why it was limited at least initially to the northeastern part of the country, this being the distribution area for this particular vehicle of infection. There were some hospital-associated cases extending into Ohio, where the vehicle of infection may also have been distributed. We have not adequately explained or explored how the Chicago hospital became involved.

DR. SLUTSKY: Has the study of the presence of S. derby been made of poultry and eggs in the West in the inner basin area between the Rockies and California?

DR. BRACHMAN: The laboratory isolation reports that are sent to us on a weekly basis have not shown any increase in S. derby isolates from that part of the country from either human or nonhuman sources.

DR. FORSTER: It is not quite true that S. derby has cropped up only in one hospital in Chicago. Our laboratory has typed 128 isolates as S. derby in the course of the past 12 months; and, of those, 72 were in one hospital, but the remainder of them were scattered over the northern two-thirds of Illinois with some not being hospital associated.

> **QUESTION:** What States other than those involved in the major S. derby outbreak have identified an unusual number of isolations of this organism?

DR. SANDERS: This question applies to the last few months. It is interesting that the States that appear to have additional hospitals involved, as late as December and January, are those that border the northeastern States originally affected.

There have been cases continuing to appear in Indiana, Illinois, and Ohio, which are in a sense contiguous with the Northeast. Just this week, I have learned of several additional cases in one hospital in Vermont. As regards the additional cases and the possibility of additional hospitals involved in Illinois, I have no idea whether the cases are hospital acquired or whether they represent the normal incidence for this period. We have not received a report on these yet. Along with this question, I wonder if Dr. Lashof, who is Chief of Preventive Medicine in a Chicago hospital, would give us a few words about her particular and somewhat peculiar problem.

DR. LASHOF: Our problem has been peculiar, and Dr. Sanders asked me to comment on it because of that. We have not shown the type of epidemiological curve which he described previously in that we have had no sharp outbreak. What we have seen is a chronic situation starting in August 1963 and continuing until the present time (March 1964) with approximately two to three new cases each week among patients hospitalized for other causes. Some weeks there are no isolations. A steady number of stool cultures have been submitted to the laboratory. We find that from August through November, 3.0 to 3.5 percent of stool cultures submitted were positive for S. derby. Throughout the month of

December and January this percentage was 2.0 percent. We frankly do not understand this steady chronic situation.

We have gone through our kitchen quite thoroughly in terms of our dietary personnel and found no cases of S. derby among the dietary personnel. We stopped feeding raw eggs and eggnog early in September. All eggs are cooked until the white is firm. Our eggs have been inspected and have all been grade A with no cracked eggs being brought into the hospital. We have obtained surface cultures in the kitchen, cultured turkey roll, and practically everything that can be referred to as a possible source of infection and have found no contamination. Our environmental cultures, including air samples, and surface samples in rooms of patients with S. derby have also been negative.

In relation to all of this, I would like to raise the question of the pathogenicity of this organism. The percentage of recoveries in our surveys of asymptomatic patients is the same as the percentage of recoveries from the routine cultures sent to the laboratory presumably from symptomatic patients. Both rates are 2.5 percent. This makes me question the relationship of this organism to symptomatic disease. If I were doing a study for a cause of a diarrhea that had not been previously identified and I found an organism in 2.5 percent of survey patients, none of you would believe that this organism caused any symptoms no matter how good it looked on the chart of any individual patient. I do not mean to imply by this that this organism never causes disease. I personnally am convinced that several of the patients were quite ill and the illness was related to S. derby. However, I would like further information on what other factors determine whether the organism causes disease or not, such as dose or state of the host.

DR. COCKBURN: It is probably true that you can have a salmonella running through a community without causing very much disturbance of any kind. Why it should do so, I do not know. The recovery of salmonella with the same frequency from both cases and controls does not rule out the possibility that the organism is causing some illness.

DR. BRACHMAN: I would like to ask Dr. Cockburn whether he would care to speculate as to why in hospital-associated infections due to S. derby, we have the problem of persistence and the evident lack of effect from appropriate control measures; whereas with other hospital-associated infections, control measures are usually effective, or at least the epidemic appears to be self limiting.

DR. COCKBURN: It is very difficult to say why this happens. All our experience in the United Kingdom suggests that it is extraordinarily difficult to apply adequate control measures for the E. coli and salmonellae. Rogers (7) in Birmingham made very careful studies on E. coli infection in children which showed that the organism was everywhere, not only in the places where you would expect it to be — such as on the towels, toothbrushes and so on — but in the floor polish and the vacuum cleaners, as well as in other unusual places.

I suspect that one of the fundamental difficulties about controlling something like an E. coli outbreak or a mild salmonella outbreak is simply that the organism is in places where you do not expect it. Also, we must look at this question of dosage again because certainly in young children and debilitated adults and probably in adults who have gastrointestinal illness of various kinds, my growing impression is that a few organisms are enough to infect and possibly to cause symptoms. In other words, I think your efforts to control it are not adequate.

DR. BRACHMAN: Concerning your first comment, is there any evidence to indicate that the organisms recovered from fomites have anything to do with the spread of disease?

DR. COCKBURN: We do not know if an organism that you get out of the dust is likely to cause illness. This not only applies to salmonella, but also to staphylococci and streptococci. Also, we do not know whether or not the organism which you culture, even if you only subculture it once or twice, is the same as the organism that you get out of the patient.

DR. BRACHMAN: Perry et al. (8) reported the recovery of streptococci from blankets used by patients with streptococcal pharyngitis. He found that other patients who had been exposed to the contaminated blankets were colonized with the organism but showed no evidence of disease.

DR. WISE: I want to compare some of the information we are talking about regarding salmonella, to the problem that exists with staphylococcus. Our rate of staphylococcal infections in hospitals ranges from a low of about 1.0 percent, in those hospitals in which there are great efforts to control the disease, up to a high of 35 percent in other hospitals. The problem becomes one of definition of infection. What is infection? We are talking about salmonella as if anyone who harbors the organism is infected; whereas in staphylococcus we cannot possibly do that because when we cultured all of the people who entered our operating theatre in a period of one week we found that 80 percent of over 200 people were harboring Staphylococcus aureus and many of them were harboring the strain that we knew was causing infection, yet we did not consider them to be infected. Consequently, we had to revise our concept. I like the phrase I heard yesterday which somebody made, that "the carriers are the victims of the environment." Therefore, if we could do something about the environment that will prevent the acquisition of carriage, this would be much better than trying to do something to the carrier.

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CONTROL OF THE SALMONELLA PROBLEM

MODERATOR: ***Dr. Archie L. Gray***

Meat Industry's Responsibilities

Charles F. Niven, Jr., Ph.D.[1]

Since salmonellae are known to be human and animal pathogens, it is not surprising that meats and meat by-products frequently contain these microorganisms. It is not the intent of this discussion to review the literature on the prevalence of salmonellae in and on meats intended for human consumption or to present evidence as to the significance of their presence. Rather, it is assumed that salmonella contamination in foods and feeds is undesirable and that all attempts should be made to minimize their occurrence.

Several excellent publications have appeared, one very recently (1) on the epidemiology of salmonellosis. Each presents evidence that links the problem of human salmonellosis with contaminated foodstuffs, especially foods of animal origin. The meat industry by its very nature is in a key position to play a leading role in reducing the incidence of salmonella infection of foods that ultimately reach the consumer. However, there are many obstacles to attaining this goal, some of which appear to be formidable. Unless some scientific breakthrough occurs, which seems unlikely in the foreseeable future, we must continue to expect some salmonella infection in unprocessed red meats that are offered to the consumer. In the meantime, several practical steps can be taken which should reduce the level of contamination.

Among fresh red meats, the major problem exists with pork and veal. Both surface and interior contamination of such meats must be considered. Galton, Smith, McElrath, and Hardy (2) reported a rather high incidence of salmonella contamination on the skins of slaughtered hogs. The high incidence was attributed to fecal contamination of the carcasses after they were scalded and during the time they were being dehaired. In contrast, the skins, after being scalded but prior to being dehaired, were relatively free of salmonellae. This report dramatically points out the importance of fecal contamination from the relaxed anal muscles during the dehairing process. Subsequent treatment of the carcasses did not significantly lower the incidence of skin contamination. These results point out the need for a concerted effort to reduce or eliminate fecal contamination of the carcasses during the dehairing operation, either through a redesigning of the conventional dehairing machine or through a refinement in its use.

Surface contamination of the carcasses appears to be the major source of contamination of abbatoir equipment, and ultimate contamination of red meats. Galton, Lowery, and Hardy (3) have noted the high incidence of salmonella contamination in pork sausages.

Salmonellae can also occur in the interior of meats as they are offered to the consumer. Since the classical work of Hormaeche and Sakamendi (4), a number of investigators have confirmed the occasional presence of salmonellae in lymph nodes, edible organ meats, and muscle tissue of animals immediately after slaughter. Such occurrences indicate infection and invasion of the living animal prior to slaughter. It is to be expected that some infected animals are slaughtered without showing any evidence of illness. The elimination of all interior contamination of meats, indeed, appears to be a formidable problem confronting the meat industry. Presently, the only hope is to reduce such occurrences by following practices that are known to safeguard the health of the animals prior to slaughter.

One might question the significance of salmonella contamination of fresh red meats, especially pork and veal, since they are intended to be thoroughly cooked prior to being consumed. Whether they play a role in human health through direct or indirect contamination of other foods should be decided by the epidemiologist.

Considerable evidence is available to substantiate a marked increase in the rate of intestinal salmonella contamination of meat animals as they proceed through the regular marketing channels and to the abbatoir (2, 5, 6). Holding pens for such animals are known to be highly contaminated with salmonellae, presumably from feces of occasional infected animals or animal carriers. Sanitation and feeding practices in such pens are known to be conducive to the infection of otherwise healthy animals. It appears that in most instances the salmonellae found in the pig intestine at the time of slaughter are merely intestinal passengers, as evidenced by their low population levels (6). However, in some instances, frank subclinical infections appear to occur,

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even to the point of lymphatic invasion. In any event, those salmonella serotypes that are most commonly found in the holding pens are most likely to be the ones that will be found in and on fresh meats from the slaughtered animals.

This situation begs for a practical solution—one which would not disrupt the entire economy of animal agriculture. If these problems could be solved, there seems to be little doubt that a marked reduction in the incidence of salmonella contamination in and on fresh meats could be achieved.

In a recent study conducted in our laboratories in collaboration with a local abbatoir, the incidence of fecal salmonella contamination in swine handled under different conditions prior to slaughter was determined. A three to four fold increase in incidence was noted in those swine which were held in holding pens 24 to 48 hours prior to slaughter, as compared with those which were slaughtered immediately after receipt from the farm. The situation worsens among special swine (boars, sows, culls) which may be held in pens for several days prior to slaughter.

In contrast with fresh meats, there seems to be little or no problem of salmonella contamination in the large number of varieties of processed meat foods. Most such meat foods are thermally processed sufficiently to kill any contaminating salmonellae. Among the hundreds of processed cured meats examined in our laboratory, we have yet to find a single salmonella strain. This situation appears to be different from that found among processed meat foods in England and the rest of Europe, where many foods are prepared which are unfamiliar in the American diet, and which may not be adequately refrigerated prior to being eaten.

Another problem confronting the meat and rendering industries concerns salmonella contamination in rendered animal by-products which are intended to be used as feed ingredients. That such contamination frequently occurs is attested by a number of publications on the subject (6, 7, 8, 9). Although the significance of such contamination has been questioned, there are a few documented cases in which outbreaks of salmonellosis in farm animals have been directly traceable to feed supplies.

When detected in rendered by-products, salmonellae are invariably present in low numbers, usually one or less per gram (6). They clearly gain entrance through recontamination of the finished product, mainly through dust from the environment (10).

In most instances, recontamination can be prevented through proper plant design, good hygienic practices by the employees, and a good housekeeping program. Unfortunately, through improper design, it is not possible to accomplish these goals in some plants now in use. On the other hand, there are rendering plants in operation which have never had a salmonella problem. The production of salmonella-free rendered animal by-products should be an attainable goal for the entire industry.

In those particular instances where salmonella recontamination cannot be prevented through conventional methods, it might be necessary to resort to some terminal treatment of the finished product. Holding in heated bins at approximately 130° F. might be practiced, or the product could be terminally heated at a higher temperature for a short time just prior to leaving the plant. Table 1 presents some results of dry heat experiments on salmonella-contaminated meals. These studies were conducted in our laboratories by Oscar G. Rassmussen, Rexford Hansen, Nicholas J. Jacobs, and O. H. M. Wilder (to be published). It should be noted that the meal which was artificially contaminated with a relatively high number of Salmonella senftenberg, strain 775W (known as the most heat-resistant salmonella strain in liquid media), was successfully heat treated much more easily than those meals which were naturally contaminated. One naturally contaminated sample which was unusually heavily infected required rather severe heat treatment in order to kill all salmonellae.

Table 1.—*Dry heat resistance of salmonellae in rendered animal by-products*

Serotype		Cells/gram	Heat resistance
senftenberg 775W	(A)	2,000	S: 150° F., 15 min. D: 155° F., 15 min.
bredeney and *derby*	(N)	1	S: 170° F., 7 min. D: 180° F., 7 min.
montevideo	(N)	0.5	S: 160° F., 15 min. D: 170° F., 15 min.
montevideo	(N)	25	S: 190° F., 14 min. D: 195° F., 7 min.

A – Artificially contaminated
N – Naturally contaminated
S – Survived
D – Population reduced to below detectable levels in 10 grams of sample

Other possible terminal treatments might include fumigation (e.g. prophylene oxide) or the use of gamma irradiation (not now feasible). The incorporation of contaminated meals into pelleted feeds might be another practical method for preparing meals that are safe. Such feeds are known to be relatively free of salmonellae.

There are several methods of rendering fats from meat and bone scraps, a few of which are in the experimental stage of development. One such method employs proteolytic enzymes which decrease the severity of heat treatment required. It is hoped that any new method that is adopted will incorporate techniques that will either solve or circumvent the salmonella recontamination problem.

Because of economic pressures, there is a tendency to handle feed ingredients, indeed all feeds, in bulk form. Such practice makes it more difficult to achieve the desired goal of preparing feeds that are consistently free of salmonellae. Such practices bring the transportation systems into the picture as having to share the responsibility.

In conclusion, the meat and livestock industries have a definite role to play in the over-all salmonella problem. The responsibility begins on the livestock farm through use of good livestock management, proceeds through transportation of the livestock to the abbatoir, continues in proper handling of meats and by-product feed ingredients, and ultimately returns to the farm by way of feeds. In this cycle of events, there are several points at which the salmonella problem might be attacked in order to reduce human and animal salmonellosis. However, any success in solving the problem seems to be far beyond the horizon.

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Role of Poultry and Egg Industry

Harold M. Williams[1]

"The world will never be made safe for democracy, because democracy is a dangerous business." So says G. K. Chesterton. Living, too, is a dangerous business beset by continuing difficulties and uncertainties. But our concern in business is to proceed cheerfully amid the congenital uncertainties and dangers of life and business without being disoriented by our inability to find total solutions and total security. This was best described by George F. Kennan when, discussing "The Illusion of Security," he spoke of "that peculiar form of American extremism which holds it possible that there should be such a thing as total security and attaches overriding importance to the quest for it." So it is with our efforts to deal with the problem of salmonellosis.

We in the food industry have this vital concern—salmonella. This National Conference on Salmonellosis is evidence of the concern by the United States Public Health Service. The attendance here of representatives of the food industry, the animal feed industry, the food producer, and public health officials attests to the very real concern of the food industry and its allies with the solution or containment of this problem as well as many other problems facing us now and those now unknown which will face us in the future.

Salmonella is a problem both in human foods and in animal feeds. Salmonella can be in and on eggs, turkeys, and chickens, as well as many other foods. We in the poultry business have for a long time been diligently working on this difficulty. We are making progress in this area with the aid and guidance of governmental agencies and educational organizations.

But the problem in some ways grows more evident, more imminent, and more complex. This enlargement of our salmonella difficulty is at the same time both beneficial and disturbing. Improved surveillance and reporting sound the alarm and increase the challenge. Mass production of food and its increasingly complex distribution complicate the situation. Ever higher consumer health standards require management to initiate improved patterns of performance.

Yes, we have a problem—but primarily a "people" problem. Our primary concern is that of motivating and activating people. Our difficulty is primarily sociological rather than technical or scientific. I am sure you will agree with me that we have right here in this room today, five times over, all the technical talent needed to get the job done were we all given dictatorial authority. But who is so foolish as to trade freedom itself for freedom from the possible danger of a bad health hazard.

What then is our aim? Our objective is to get the maximum in continuous control over salmonella with a minimum of outside compulsion. Just what do we mean by compulsion from outside the poultry industry: governmental disciplinary action and regulation—Federal, State, local; press threats which destroy our markets; economic pressures by big customers. We prefer internal self-discipline and action whenever and wherever possible.

What basic precepts do we need to successfully meet the challenge? We will make these our business policy, our working philosophy:

1. We need to develop and encourage an abiding commitment of management to continually improve food processing.
2. Management needs that sophistication or that equanimity which comes only from a thorough knowledge of food technology. This balance or sophistication will help management place health issues and incidents of malfunction in their proper perspective.
3. Businessmen must have a ready willingness to work with governmental agencies and educational institutions in health matters.

This is our working philosophy: moral commitment, technical sophistication, and social willingness.

Yes, we have a problem, but we are working on it and we are making progress. A massive coordinated approach by the food industry, food and drug officials, public health authorities—both Federal and local—and consumer groups calls for adjustments in thinking by all parties concerned.

For example, it may come as somewhat of a shock to some operators in the poultry and egg business that the salmonella problem will not totally succumb to direct, immediate action. Also, a better mutual understanding between the businessman and the public health official will come about when both realize that people in government and regulatory positions are, in general, of the same caliber as people in industry. They are subject to the same frailties and weaknesses, they are not perfect, nor can we expect perfection of them. Herein lies a certain peril. A volatile situation exists when there are hundreds of public officials to deal with — Federal agencies, five States, hundreds of municipalities — all of these concentrating on the control of salmonella. The irresponsible action of just one of these persons could mean great economic loss to the industry and would not contribute to the solution of the problem at all. Occasional outbreaks, isolated facts and rumors and partial truths can be catapulted into dangerous situations by over zealous public servants. Health faddists, too, always constitute a danger.

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The consuming public, as such, is getting increased representation at the Federal level. This representation is all to the good and will benefit the consumer and the food industry if the truth is sifted from the partial truths, the isolated facts, and the misunderstandings. For example, packaging presents problems to the food manufacturer which may be little understood by the consumer or his representatives. The technology behind the package fill, the necessity for secure closures and the varied net weights should be described and brought to the attention of these interested and dedicated consumer representatives. Some of these variations actually benefit the consumer by providing a wider selection and greater convenience.

Let prudent businessmen as well as regulatory public officials pattern their thinking and build their operating policy around these two cornerstones: totality and relativity. As you well know, an isolated fact is not necessarily the truth unless related to the total situation. An isolated fact or occasional instance must be comprehended in the entirety of the situation by relating it to all the pertinent factors. For example, discretion and judgment should be exercised when interpreting results based on a specific sampling and testing procedure.

Dangers in themselves are not absolute but are relative. They are measured primarily in terms of alternative dangers — more — less. We must proceed steadily but cautiously. We must not be stampeded into ridiculous attitudes or into unwise action. But proceed we must, and together — industry, government, education — and this we are doing. We have been impressed with the generous cooperation of the staff of the Communicable Disease Center who are concerned with salmonellosis.

We appreciate your positive rather than negative approach, you reliance on leadership and education, rather than on legislation and regulation. We respect your responsibility to public interest, and we appreciate your responsibility to industry.

The following activities indicate your leadership:

1. Personal appearances on Institute programs such as the Fact Finding Conference, quality control schools, committee meetings and meetings of the Board of Directors.
2. Salmonella displays which are made available for public meetings.
3. Your training program at the Communicable Disease Center in Atlanta, Georgia.
4. Your training program at the Robert A. Taft Sanitary Engineering Center in Cincinnati, Ohio.
5. Various educational programs, statistics, reports.
6. This impressive session being presently carried on.

This massive coordinated approach on salmonellosis is most commendable:

Food and Drug - Supervision
Communicable Disease Center - Surveillance and Reporting
United States Department of Agriculture - Inspection
USDA Western Regional Research Laboratory - Research Work

Yes, we are moving together. The Institute of American Poultry Industries, too, has been pressing forward. Our Research Council's Microbiology Division, composed of industry bacteriologists, compiled 11 methods in use throughout the world. Just recently, collaborative studies for the testing of salmonella have been carried on. As you may have noted from the Communicable Disease Center's Salmonella Surveillance Report No. 20 of January 6, 1964, the Institute's Research Council has been working diligently in this area. Our Council's recommendations on salmonella were reported in this surveillance report. The Institute continues to carry on a series of quality control schools to train industry members to do a better quality control job. Educational sessions in our poultry, shell egg, egg products, and sanitation committees are continuously conducted. Representatives of Food and Drug and industry have reviewed the salmonella problem before various meetings of our Board of Directors.

Yes, the Institute has been working with other phases of the industry in a coordinated program. And, ladies and gentlemen, we in industry value free enterprise, but we must never forget that the vitality, the strength of free enterprise rests upon serving public interest. Yes, public interest must have priority over economic interest.

What must the businessman keep in mind when serving public interest in the area of public health? He must have a constant alertness to dangers, an understanding of the gravity of the situation and the urgency of health hazards, and the ability to marshall and utilize available knowledge and resources in combating salmonella. And then through industry trade associations the businessman can coordinate industry-wide efforts with education and government.

Yes! We in industry are moving in the area of health issues. We have been and will continue to move forward.

Role of Animal Feed Industry

Charles F. Rossow, D.V.M.[1] *and*
Charles W. Darby, D.V.M.[2]

The magnitude and scope of the salmonella problem has been presented during the first two days of this conference. It has been established that salmonella serotypes can be isolated from some commercial feeds as well as selected feed ingredients.

Excluding some of the more or less specific salmonellosis infections, such as pullorum disease, fowl typhoid, and the more common paratyphoid infections of livestock and poultry, the importance of almost a thousand salmonella serotypes has not been established. We, as a member of the animal feed industry, are and have been very much interested in the practical aspects of the over-all problem. Active research programs and projects are being directed toward establishing not only the significance of salmonella contamination but also such things as the following:

1. The significance of minimal numbers of organisms in either feed ingredients or mixed feeds. Several reports have shown that the number of salmonella organisms recovered from a contaminated sample is low. The figures presented ranged from 2 to 200 organisms per gram of contaminated feed. Are these levels meaningful?
2. The pathogenicity of salmonella serotypes isolated from feed and feedstuffs.
3. The role of medicated feeds in the control of minimal numbers of contaminating organisms in feeds as they are consumed by poultry and livestock.
4. The importance of other sources of contamination such as wild birds, rodents, and domestic animals.

Again, it is not within the province of this paper to review the complexities of salmonellosis or to establish any alibis for the feed industry. On the contrary, the feed industry is proud of its accomplishments in this field as well as in others. Salmonellosis is not a new phenomenon. It has been with us since the beginning of the feed industry. Improvements in feed processing methods and techniques, more stringent quality control programs, better production equipment, and more knowledge about the disease itself, have all combined to keep the threat of salmonellosis well under control. The entire environment of today's feed industry is more favorable for less contamination than ever before. Improved techniques for the isolation and identification of salmonella bring this matter more to the public attention and at the same time allow even better controls to be put into practice. We believe that the entire salmonella matter should be considered as just one of the many infectious diseases that must be controlled to maintain healthy livestock and poultry and protect the public health. With the increasing number of isolations and identification of new species, there is definitely a need for a simplification of its control.

Although we are cognizant of the importance of salmonella contamination in feed and feedstuffs, teamwork between all phases of the animal feed industry will minimize its effects. Despite the broad aspects and many of its ramifications, we will attempt to present some of the practical procedures and programs that have been instituted by the animal feed industry as an aid in the over-all control of salmonella contamination.

Following preliminary surveys of feedstuffs and feed ingredients conducted in our laboratories, it soon became evident that meat scraps, fish meal, and other meat by-products were the principle sources of contamination. However, these are by no means the only sources of salmonella, for grains and other feed ingredients have also been involved.

As part of an over-all control program, the feed industry is attempting to evaluate and consequently select good sources of meat scraps, bone meal, poultry by-products, fish meal, and other related products, that are free of salmonella. It has been established that these products are initially prepared under conditions that would ensure their freedom from salmonella organisms; from this point, the problem is one of recontamination. We have been working with our buyers and suppliers to upgrade the source of these ingredients in an attempt to eliminate the possibility of recontamination. Here again, producing feedstuffs under practical economic conditions makes this very difficult. Major steps have been taken by the United States Department of Agriculture to establish minimal requirements for plant sanitation and product handling in order to supply a salmonella-free product.

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Another very important factor to consider in this chain of quality control rests in the transportation of the by-products to their ultimate destination in the feed mill. Unless precautions are taken for the cleaning and disinfection or possibly even fumigation of common carriers, the procedures outlined above would be of very little practical value.

The next possible source of contamination would be in the feed mill itself. Today the feed industry is attempting to establish plant sanitation codes and programs to eliminate the possibilities of contaminating feeds. These programs will vary from plant to plant, but they very definitely must be a very important part of the over-all quality control procedures of the mill. In our organization, we have attempted to institute a sanitation program that parallels the guide lines recently suggested by the subcommittee on contamination and recontamination of by-products. We have developed a program which covers the following:

1. General aspects of the problem including the control of wild birds, pets, and rodents as well as a good housekeeping program for the surrounding building and grounds;
2. The type of building construction and facilities to be used in order to maintain clean buildings;
3. Employee facilities and requirements;
4. Moisture and dust control within the plant;
5. The handling of used bags of all types and the preparation of trucks and cars for bulk shipments;
6. The bacteriological examination of feed ingredients and finished feedstuffs;
7. Responsibility and training program.

After it is manufactured, the feed may go directly or indirectly to the customer. It may be transported in bags or bulk. In somes cases, there would be a direct line from the feed mill to the consumer: for example, a large poultryman might bring his own bulk trucks directly to the mill, and these in turn would be used to transport the finished feed to his own bulk tanks or feeders. Under these conditions, it would be the responsibility of the consumer to guard the quality of the product after it leaves the mill.

Another direction of manufacturing involves the production of concentrates by the mill. These in turn are used to produce complete feed by the local feed dealer or small manufacturer. Under these conditions, the local dealer or manufacturer would be responsible for continuing the bacterial quality of the final feed mix. This actually means the local dealer would have to provide the same production control program as the mills.

It is obvious from the above discussion that, through rigid quality control programs, we can minimize possible salmonella contamination of animal and poultry feeds. Since it is virtually impossible to examine every feedstuff on a routine basis for the presence of salmonella, the only other possible solution to the problem is to process the feed in such a way as to render it free of salmonella and the other common bacterial disease-producing agents. There are several different ways this could possibly be approached. Again, the economics of these methods preclude their use for poultry and livestock farm operations.

Irradiation has been used on an experimental basis but has some very obvious shortcomings. It should be kept in mind that the "pasteurizing process" must also be compatible with the essential feed ingredients of the rations involved. It has been our experience that selective irradiation does eliminate all pathogenic bacteria; but, at present, it is not a practical solution. At the present time, the costs involved in irradiation and the relatively small volume would eliminate its use in all but experimental diets.

Within the past few years, a process which is somewhat similar to commercial pelleting has gained wide attention and acceptance. This is the process of extrusion, which is capable of conditioning the product to higher temperatures and moisture levels than the common pellet mill. As a result of these facts, salmonella contamination has been eliminated in properly extruded products.

We have reviewed several conflicting reports concerning the possibility of the pelleting process killing salmonella organisms in feeds. In a series of research tests conducted in our own laboratories and pilot plant operations, we have determined that the conventional ring-type die pellet mill reduces, but does not eliminate all salmonellae from the pelleted feed. Table 1 presents the results obtained from cooperative projects carried out by our engineering and bacteriology departments.

As a word of caution, we would like to point out

Table 1.–*Expansion extrusion compared to pellet extrusion in killing salmonellae*

Sample	Number of samples	Temperature	Time (seconds)	Moisture (percent)	Salmonella positive
Expansion extruded feeds[1]	775	200°-350° F.	45-60. . .	25-35 . .	0
Pelleted extruded feeds[2].	35	160°-180° F.	2-16. . .	11-19 . .	21

[1] 10% of mash feed samples contaminated with from 50 to 80 million *Salmonella* sp. per gram. [2] All mash samples contaminated as in above.

that producing salmonella-free feeds is only the first step in the control of the over-all program. Great care and expense would then have to be invested to protect the finished feed until it reaches the ultimate consumer.

In summary, it should be restated that salmonella contamination of some feed ingredients and finished feeds can be very complex and economically difficult to control. We do not wish to minimize the importance of salmonella nor of the responsibilities of the feed industry. We have pointed out that the feed industry has recognized the importance of the over-all conditions and has definitely taken steps towards minimizing contamination. A strict quality control program can and does reduce the amount of contamination under today's conditions of manufacture. The present day pelleting process undoubtedly reduces the bacterial contamination of feeds, but does not ensure complete "pasteurization" of the feeds that would result in a consistent salmonella-free product. Cooperative experiments with the engineering and microbiology departments have shown that with pilot production facilities a satisfactory "pasteurization" process can be worked out for small quantity, high cost experimental diets. Diets of this type for laboratory animals are currently commercially available.

We are still concerned about the practical aspects of handling these "pasteurized" feeds following manufacture. At the present time, only producers handling gnotobiotic animals could take full advantage of them.

As we review the facts pertaining to salmonella, it should be kept in mind that the feed industry has kept pace with modern technology. It is doing a better job today than ever before in helping farmers produce high quality, wholesome meat, milk, and eggs at prices within the reach of all consumers.

Role of Food and Drug Administration

Glenn G. Slocum, Ph.D.[1]

The Food and Drug Administration quite obviously has a vital role in the control of the salmonella problem resulting from dissemination by foods in interstate commerce. The Federal Food, Drug, and Cosmetic Act defines the term food as ". . . articles used as food or drink for man and other animals;. . . and articles used for components of any such article." It defines a food as adulterated if it contains a poisonous or deleterious substance which may render it injurious to health; if it has been prepared, packed, or held under insanitary conditions whereby it may have become contaminated with filth, or whereby it may have been rendered injurious to health; or if it is in whole or in part, the product of a diseased animal or of an animal which has died otherwise than by slaughter. For sometime, FDA has held that interstate and imported foods containing salmonellae are adulterated and subject to the legal sanctions of the law.

Although the primary responsibility for the wholesomeness of meat and poultry products resides in the inspection agencies of the U. S. Department of Agriculture, the Food, Drug, and Cosmetic Act can be used as an instrument for the removal of dangerous products from interstate commerce, once they have left inspected plants.

The Act also provides that the Secretary may cause to be disseminated information regarding food, drugs, devices, and cosmetics in situations involving, in the opinion of the Secretary, imminent danger to health, or gross deception of the consumer. The statute, therefore, provides a useful instrument for the control of foods which serve as vehicles for dissemination of salmonella.

FDA operates through a network of 18 field districts located in principal cities throughout the country, augmented by resident inspection of posts in many other cities. About 900 inspectors are currently available for the massive job of inspecting food and drug establishments to detect potential violations of the law, to collect samples, and to investigate injuries and trace suspected products. While not trained specifically in epidemiology, the inspectors invariably hold college degrees in some branch of science, and are keen and intelligent observers. They constitute a unique and skilled force in tracing the distribution of products, and, where necessary, they aid in securing the removal of products from the market. They constitute, with their counterparts in State and local food and drug agencies, a tremendous resource for prompt action once a hazardous situation is identified.

In recent years, a program of equipping and staffing the field districts for bacteriological activities has been accelerated. Some 20 bacteriologists are now located in 12 district laboratories, and this staff will be augmented as rapidly as possible to include each of the 18 districts. While still inadequate to cope with the increasing work loads in the field of microbiology, they already constitute a valuable FDA resource to deal with specific problems, such as the detection of salmonellae in samples of imported coconut and egg products collected during field operations. A small group of microbiologists in the Division laboratories in Washington is engaged in research on salmonellae, in the development of methods for the detection of salmonellae in foods, and the serological identification of strains isolated. In addition, we can provide analytical services to Districts not presently supplied with bacteriological facilities.

The resources available to the Food and Drug Administration are substantial and can contribute significantly to control of the salmonella problem. We are best equipped to deal with those problems which are clearly identified and delineated, e.g. the detection of contaminated lots and the recall of foods identified as the causes of outbreaks or cases of salmonellosis. In addition to such actions, control programs have been undertaken on two commodities. The implication of dried coconut as the source of outbreaks of salmonellosis in other countries led us to institute a program of testing shipments of the products offered for entry into this country. A distinct problem was revealed; and, as a result of changes instituted by the industry, salmonellae have been virtually eliminated from products offered for entry. Under a program initiated last year, regulatory actions are being undertaken to eliminate traffic in egg products and other foods containing egg products which contain salmonellae.

We have also considered and, to a limited extent, employed routine tests for salmonellae in foods collected during sanitary inspections of food establishments. Such tests generally have not been productive; and we believe that, for the wide range and volume of foods in interstate commerce, they are impracticable with our present facilities.

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It must be recognized that the Food and Drug Administration is in a poor position with respect to the initial discovery of outbreaks or cases of foodborne salmonellosis or other foodborne disease. We are largely dependent upon other public health agencies to provide the primary leads which indicate or identify specific problems of food transmission. In order to bring our resources to bear on these problems, we urge and encourage the cooperation of other agencies in early notification of FDA in situations potentially involving interstate foods.

Interstate foods have been implicated in a few outbreaks of salmonellosis during the past decade and have been suspected of implication in widespread outbreaks caused by *Salmonella typhi*, *S. reading*, and *S. hartford* during the same period. We are concerned with the apparent increase in the incidence of sporadic cases of salmonellosis, which may be symptomatic of foodborne outbreaks unrecognized because of failure to connect them with other cases. Specific identification of such foods and their sources is essential to progress in the prevention of foodborne salmonellosis.

It is quite evident that we need improved systems for the investigation, detection, and reporting of food-borne salmonellosis as a guide to the application of necessary control measures not only by the Food and Drug Administration but by all governmental agencies and the affected industries.

Role and Activities of the U.S. Department of Agriculture

Merton R. Clarkson, D.V.M.[1]

The new look we are taking this week at a very old public health problem is giving us new insights into the mounting importance of salmonellosis.

How can we control groups of organisms that are so diverse, so widespread, and so pervasive; that have such different means of survival; and that attack man and so many other animals?

Of the hundreds of types of salmonella organisms found, relatively few commonly cause illness in man. However, under certain circumstances, any species of salmonella must be considered potentially capable of producing disease in any animal.

In the case of each of the salmonellae, we must find a vulnerable point and strike there to cut the chain of disease transmission—a chain in which animals, materials, and man may all be links.

We often assume that the transmission cycle of salmonella organisms moves from animals and animal products to man. I would like to remind you that sometimes this cycle may be reversed.

The importance of man in transmitting salmonella to other animals must not be underestimated. Each year, cases are reported that incriminate human carriers as the source of infection for animals. How often man is responsible for outbreaks among animals is not clearly known, partly because of gaps in reporting procedures and inadequate reports from a small number of States.

Recent cases in Germany strongly suggest a human-to-animal transmission. In Poland, man has frequently been recognized as the carrier of types of salmonella organisms before they could be recovered from livestock.

I believe it is important for us to study and better understand the mode of disease transmission from man to animal, as well as from animal to man, in order to gain a deeper insight into the many complex ways that disease can spread in living organisms.

The occurrence of salmonellae in American agricultural products has been of concern to the United States Department of Agriculture (USDA) since these bacteria were first isolated.

As most of you know, one of the first men to work with salmonella organisms was Dr. D. E. Salmon, the first chief of the Department's livestock research and regulatory work. The year was 1885, and Dr. Salmon then thought he had isolated the cause of hog cholera instead of the associated organism it proved to be. The generic name Dr. Salmon gave his type species, the first paratyphoid bacterium to be recognized, was later changed to salmonella in honor of his work with this group.

Although the amount of work the USDA devotes exclusively to salmonellosis today is limited, a great deal of our research and regulatory work is aimed at reducing the incidence of harmful bacteria in animals, poultry, and food. I am convinced that, in the current state of our knowledge, our best protection against salmonellosis in both man and animals lies in two areas: in attention to the health of our livestock and poultry, and in good sanitation.

In the face of mounting numbers and greater concentration of domestic animals and birds on farms, in transportation, and in commerce, livestock health is being steadily improved. This is particularly a safeguard against salmonellosis, because salmonellae are opportunists, waiting to strike other animals as well as humans when they are most vulnerable—in infancy, in illness, and in old age. Research has improved animal husbandry practices that directly affect animal health—the increased automation of livestock handling, for example, and the proper disposal of animal wastes.

Research has made possible successful campaigns against animal diseases. The cooking of garbage fed to hogs, which helped to eradicate vesicular exanthema from our swine herds and is doing the same for hog cholera, also destroys salmonellae and many other disease organisms. The animal inspectors at our ports and borders who guard against the importation of unhealthy animals and contaminated animal products are another protection to livestock health.

We can never overemphasize the importance of sanitation in combating salmonellosis—sanitation on the farm, at animal concentration points, in industry, and in the home. Salmonella organisms are destroyed readily by the usual disinfectants, but they may survive for long periods of time in soil, manure, and sheltered places.

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One of the Department of Agriculture's basic responsibilities in sanitation is to make sure that the bactericides on the market will do their job efficiently and safely. USDA regulates all products sold interstate that claim bacteria-destroying properties, except substances used on living creatures. We have registered about 7,000 such materials. These include the germ-fighting chemicals used on farms and in water supplies, hospitals, restuarants, food processing plants, beauty shops, barber shops, and homes.

The primary test organisms used in determining the efficacy of germicides and disinfectants are two salmonella species, Salmonella typhi and S. cholerae-suis. No product is accepted for general use unless it is effective against these and other organisms.

In dealing with salmonellosis, we endeavor to marshall the full talents of USDA in research, regulatory, and educational disciplines. Veterinarians, chemists, bacteriologists, epidemiologists, nutritionists, marketing and extension specialists, and many others may participate in this team approach.

We also work closely with the industries involved, with State and other Federal agencies, and with other public and private institutions and groups.

The salmonella organisms are widespread. They are found in cattle, hogs, sheep, and horses as well as in many other domestic and wild animals and fowl. Salmonellae can infect eggs and raw milk and contaminate animal feeds, meat, and dressed poultry.

Salmonellosis is probably the most important of the avian diseases that are transmissible to man. The paratyphoids, which can infect practically all animal species, including man, cause severe losses among young fowl.

State agricultural workers have done a great deal to combat salmonellosis. It is hoped that States with the facilities and personnel to do so will step up their investigations of this widespread problem. Many States are operating testing services to detect flocks of poultry infected with S. typhimurium, one of the paratyphoid species. We have only scratched the surface here, and much more work is needed on the infection this organism causes in humans, poultry, and livestock.

The means to control two other salmonella diseases of poultry, pullorum and fowl typhoid, were developed through research in the 1930's and 1940's. Our cooperative poultry improvement programs, the most complete in the world, have reduced pullorum incidence to the lowest in the world and have practically eliminated it from turkeys. Also, the incidence of fowl typhoid has been drastically reduced. The practicality of a concerted drive to eradicate these two diseases from all our domestic flocks is now being discussed. Although these are essentially diseases of poultry, their organisms have been known to produce disease in humans.

Generally speaking, our chief problem is not that unhealthy animals and birds reach the market. The information available to us indicates that contamination may be the chief problem.

In some cases, contamination of meat and poultry can occur during processing. The danger of such contamination underscores the necessity for the strict sanitation requirements of Federal poultry and meat inspection. These systems are the most rigid, and the finest, in the world. Even before a meat or poultry processing plant is built, the blueprint must show that Federal requirements for sanitary operation can be met. Such control continues from slaughter of the animals and birds—and particularly during dressing—until the finished product leaves the plant. All these safeguards are necessary to assure the consumer that federally inspected meat and poultry are safe and wholesome.

Salmonellae may also be present in egg products. In efforts to improve the utilization of eggs and poultry, our scientists are trying to learn more about how salmonellae get into egg products. They have developed methods for detecting and counting such species in egg products. They are determining the heat sensitivity of different species and strains of salmonella as a basis for developing better pasteurization methods and are evaluating the efficiency and practicality of chemical and heating methods.

At present, the pasteurization of whole egg and yolk products is effective if properly done. Egg white, which is more sensitive, has not in the past been satisfactorily pasteurized in liquid form by heat. However, we have recently obtained exciting results which make it appear that this problem has been solved. We are pushing ahead rapidly with this research.

Our home economists prepare bulletins and demonstrations to show people how to cook dried eggs properly. The scientists have used dried whole egg solids successfully for scrambling and in baking under certain conditions. They found that baked foods that reach a temperature of 160° F. or higher can be considered safe from any salmonella organisms that might be present in the food ingredients.

They recommend that no more than six-egg quantities be scrambled on top of the stove. The eggs must be thoroughly cooked. Larger quantities of dried eggs require longer cooking in the oven.

Our publications emphasize the need for cooking all foods containing animal products long enough to destroy ordinary pathogenic bacteria. We do research in the Department and under contract to determine safe methods of cooking poultry and other foods.

Much has been said and written about the contamination or recontamination of animal feeds, meat and poultry by-products, and fish products by salmonellae and other organisms capable of producing disease. The Department is deeply concerned with this possibility. Only limited epidemiological evidence is available for us to evaluate the extent and significance of such contamination.

In order to obtain reliable data and to aid in correcting this situation, we have developed, in cooperation with others, a recommended procedure for isolating salmonella organisms from animal feeds and meat products. Isolation of such organisms, and their typing at the National Animal Disease Laboratory at Ames, Iowa, which Dr. Van Houweling discussed previously, are of course the first steps in controlling any contamination.

Sets of "Sanitation Guidelines" for the industries concerned have also been worked out cooperatively. They outline practical steps considered necessary to produce some feed ingredients that are free of salmonella organisms. The guidelines will be publicly distributed soon.

The Department is now studying feed contamination and its effect on animal health, as part of a continuing evaluation of the entire salmonella problem. Epidemiologists are working with State officials and industry groups in a three-point program.

First, they are maintaining and coordinating a continuing, nationwide study and analysis of the role that animals, poultry, feed ingredients, and complete feeds play in salmonella transmission.

They will study disease outbreaks in animals and poultry on farms. They will not overlook the opportunities for animal infection at sale barns and livestock concentration centers. They will go into problem areas in industrial plants and trade channels.

Second, steps are being taken to establish a surveillance system for checking feed components and complete feeds.

And third, where problems are identified, the Department will cooperate with State authorities and industry groups in correcting them. Various industry groups are adopting management procedures that are designed to prevent feed contamination.

These are some of the things we in agriculture are doing now about salmonellosis. What can we do in the future to protect man and his animals against this disease?

Here is agriculture's approach.

Our first concern in protecting consumers must continue to be the improvement of general health and sanitation of our poultry and livestock.

We must also explore the gray areas of our knowledge about salmonellosis through research, through surveys, and by continued surveillance of potential trouble spots.

The knowledge we gain must then be translated into action programs as quickly as possible.

In addition, we should extend the protection afforded by Federal inspection standards to cover all meat and poultry sold in this country — intrastate as well as interstate. Last month, in President Johnson's message to Congress on consumer interests, he recommended legislation to accomplish this.

And last, we must bring sound information continuously and concisely to the attention of those who are in a position to do something about it, whether they be on the farm, in industry, or in the home. The handlers of food in public facilities are subject to regulation and surveillance; but, with American homemakers, we can use only education and persuasion.

In as many ways as possible, we in agriculture shall stress over and over the proper handling of foods of animal origin. We shall urge homemakers to make sure that they handle these foods in a sanitary manner, that storage is cold enough, and that cooking methods are hot enough and prolonged enough to prevent or destroy bacterial contamination.

Role of the Division of Environmental Engineering and Food Protection - PHS

Keith H. Lewis, Ph.D.[1] *and*

Robert Angelotti, Ph.D.[2]

The Division of Environmental Engineering and Food Protection is a part of the environmental health component of the Bureau of State Services. It consists of six branches: Milk and Food, Shellfish Sanitation, Interstate Carrier, Research Grants, Special Engineering Services, and Metropolitan Planning. Each branch has a role in assisting other governmental agencies at the Federal, State, and local levels, as well as industry, in the development and application of sanitary measures for the prevention and control of milk-, food-, and water-borne diseases, including salmonellosis. In addition, the Interstate Carrier Branch has legal responsibility for enforcement of the Interstate Quarantine Regulations pertaining to the sanitation of airlines, buses, railroads, ships, and other public conveyances.

Generally speaking, the Division's activities may be grouped in the three categories of research, technical assistance, and specialized training. The headquarters staff in Washington, D. C., is supported by representatives in each of the nine Regional Offices of the U. S. Department of Health, Education, and Welfare, by research and training specialists at the Robert A. Taft Sanitary Engineering Center in Cincinnati, Ohio, and by the personnel of three regional shellfish sanitation research laboratories at Purdy, Washington; Dauphin Island, Alabama; and Narragansett, Rhode Island. The more important contributions of the branches to the prevention of salmonellosis are described briefly below.

MILK AND FOOD

Intramural research on the occurrence of salmonellae in foods and the prevention of food-borne salmonellosis has been conducted for several years by the laboratories of the Milk and Food Branch at the Robert A. Taft Sanitary Engineering Center, Cincinnati, Ohio. These activities include surveys of market products, comparative evaluations of laboratory methods for the detection and enumeration of salmonellae in foods, and investigations of selected food-borne outbreaks of salmonellosis. For example, Wilson et al. (1), found that approximately 17 percent of the raw poultry obtained from Cincinnati retail markets contained salmonellae. Additionally, 4 percent of pork, 3 percent of lamb, and 1 percent of beef specimens were positive for salmonellae. Studies of this type are designed primarily to determine the distribution and prevalence of salmonella contamination in foods, but they also help the research staff maintain its competence in evaluating newly developed methods and assist the Branch in recommending additional sanitary measures to other governmental agencies and the food industries. Though such activities are at present rather limited, additional surveys of product contamination are comtemplated as a continuing source of information about developing trends of salmonella contamination and new problems resulting from technological changes in food production, processing, packaging, distribution, and serving.

Members of the Milk and Food Research staff are currently working with several national and international organizations — including the National Academy of Sciences, the Association of Food and Drug Officials of the United States, and the Standing International Committee on Microbiological Specifications for Foods (2) — to determine whether microbiological criteria for foods may be useful in reducing health hazards from salmonellae as well as from other food-poisoning organisms. Technical assistance is also offered to various trade organizations concerned with sanitary production of commercial egg products, food mixes, bakery goods, prepared meals, and the like. Relating contamination of the final product to specific raw materials and commercial practices will help improve sanitation and thus significantly reduce occurrence of food-borne salmonellosis.

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Comparative evaluations of methods for the recovery of salmonellae have been undertaken to determine the sensitivity, reliability, and quantitative nature of various proposed techniques. These studies have been designed to evaluate the effects of food constituents, contamination levels, and processing or storage conditions on the recovery of salmonellae from food under specific conditions of incubation, media formulation, and selective or nonselective pre-enrichment. This type of study eventually culminates in the selection of methods to be used and recommended by the Public Health Service for the examination of foods (3, 4). Contributions to the latter were made by staff members of both the Robert A. Taft Sanitary Engineering Center and the Communicable Disease Center.

Other studies have been directed toward developing a body of information about the effects of time and temperature on the growth, survival, and destruction of salmonellae in foods (5, 6). These studies were designed to gather data relative to the ecological factors that alter the temperature responses of salmonellae in food. Information of this type has proved invaluable to environmental health workers who are concerned with establishing safe minimal and maximal conditions for storage and serving of potentially hazardous foods.

The Milk and Food Research section assists in the laboratory investigation of outbreaks of salmonellosis on a selective basis, as, for example, when food served aboard an interstate carrier is suspected of being implicated in an outbreak. Requests for assistance from State and local health departments are also accepted when the circumstances indicate that the findings may be useful in our on-going research projects. Although resources are not available to support a routine service, experience indicates that food specimens from outbreaks often provide the research staff with valuable materials for such related studies as methods evaluations. Additionally, the thorough examination of specimens in the research laboratory may allow unusual situations to be recognized. One such occurrence was reported by Angelotti et al. (7). This is the first paper published on an outbreak of salmonellosis from ham in the United States. Experience in laboratory investigations of outbreaks also gives the research worker better insight and understanding of the problems encountered by personnel in public health laboratories and helps the investigator make practical constructive suggestions.

The Milk and Food Branch also advises health department personnel on the establishment and maintenance of programs on milk and food protection within the States. It works closely with numerous groups such as the 3-A Standards Organization, the Interstate Milk Shipment Conference, and the National Restaurant Association, as well as several official agencies, including the Food and Drug Administration (FDA), the Department of Agriculture (USDA), and the Fish and Wildlife Service (FWS), to improve milk and food equipment, processes, and products. The Branch also develops and publishes guides on recommended sanitation practices, such as the Milk Ordinance and Code (8), Food Service Sanitation Manual (9), Vending of Foods and Beverages (10), and the Poultry Ordinance (11). Widespread utilization of these recommendations by State and local health agencies is an important factor in the control of milk- and food-borne disease transmission. These activities, though not designed for the sole purpose of reducing the extent of salmonella contamination in milk and food, have accomplished much toward this end.

SHELLFISH SANITATION

The primary interests and responsibilities of the Shellfish Sanitation Branch are centered on the prevention of disease transmission through oysters, clams, and mussels, which are potential vectors of salmonellosis, infectious hepatitis, paralytic shellfish poisoning, and the like. During the feeding process, they accumulate bacteria, as well as plankton and other particulate food, from the water. Consequently, if living in a polluted environment, they take in whatever pathogenic organisms are present. Since the entire animal is consumed, often raw, shellfish can transmit salmonellae and other enteric pathogens directly from water to man.

Public health protection is accomplished by control of shellfish-growing areas. Through sanitary surveys, safe areas are delineated; harvesting from polluted areas is prevented by patrol and depletion of natural stocks in such unsafe areas. Before development of a nationwide program of sanitary control, shellfish were involved in numerous outbreaks of typhoid fever; but in recent years, typhoid outbreaks have been reduced to a few widely scattered incidents, usually traceable to surreptitious operations by individuals who harvested from polluted areas.

Protection of shellfish-growing areas is becoming increasingly more difficult, with the population in coastal areas increasing at a rate far exceeding that in inland areas. Contamination from agricultural sources is not to be overlooked, in view of the demonstrated presence of salmonellae in the discharges of poultry and other farm animals. Solution of the problem is two-pronged. Pollution control is of course the first line of defense; but, to protect against chance contamination, it is inevitable that shellfish from certain areas, at least, will have to be depurated through utilization of their feeding—cleaning mechanism or other processes before consumption.

The shellfish sanitation program also extends into the areas of processing, storage, and distribution. The Public Health Service, in cooperation with the States and industry, has developed a manual of recommended practices for the Sanitary Control of the Shellfish Industry (12). This manual is a guide to the design, equipping, and operation of shellfish-processing establishments, and to methods for handling the product while in commercial channels. The control program is augmented by an extensive research program carried out in research centers located on the Atlantic coast, the Gulf coast, and in the Pacific Northwest.

Problems also exist in the handling of other marine foods. They may be contaminated at the source or during processing. Notably, in the processing of fish meal, raw fish from an unpolluted ocean environment may be exposed during processing to situations that result in contamination and possibly growth of salmonella organisms (13).

INTERSTATE CARRIER

The Interstate Carrier Branch of the Division is responsible for enforcing the provisions of the Interstate Quarantine Regulations applicable to water supply, waste disposal, and food service on airlines, vessels, railroads, and buses.

Water for potable or culinary purposes loaded onto any of these conveyances must meet the Public Health Service Drinking Water Standards (14). State health departments are requested to provide data on the bacteriological and chemical quality and physical characteristics of 760 public water supplies in the United States. From these, a list of approved supplies and watering points is published annually for use by carriers.

Milk and fluid milk products used on carriers must be pasteurized, and frozen desserts must be manufactured from pasteurized milk. These products must be obtained from approved sources; a list of these sources is also published annually. Shellfish served on any vessel, dining car, or airplane must be from a dealer currently listed by the Public Health Service as holding an unexpired and unrevoked certificate issued by a State authority.

A field staff of more than 30 sanitary engineers and sanitation specialists is continually involved in inspecting water supplies, watering points, and milk sources; they board vessels and dining cars and visit airline catering establishments to see that safe, sanitary procedures are followed in the storage, preparation, and serving of food.

Whether an illness was caused by contaminated food served on an interstate carrier is difficult to ascertain. Only when victims are members of an organized group on excursion or tour can accurate and complete evidence lead to a definite conclusion. The thousands of independent travelers who are served meals daily on carriers are, in most cases, dispersed within a few hours after consuming the food, milk, and water. As a result, no proved outbreak of salmonellosis has been connected with the operation of an interstate carrier.

RESEARCH GRANTS

The Research Grants Branch of the Division is presently supporting 18 extramural research projects to investigate various facets of the salmonella problem. To date, more than one million dollars has been allocated to these projects, and an additional quarter million has been committed for their continuation. These studies are concerned with such subjects as methods for the destruction of salmonellae in eggs; the effects of naturally occurring microbial inhibitors (such as anthocyanins) on the growth of salmonellae in foods; the persistence of enteric pathogens in frozen seafoods; the radiation resistance of salmonellae in fish; surveys of market foods for salmonellae and other enteric bacteria; the prevalence, persistence, and growth of salmonellae in wrapped sandwiches; the influence of antibiotic feeding of laying hens on salmonella infection in poultry and fertile eggs; and the incidence of salmonella contamination of the oriental foods commonly consumed in Hawaii. In addition, a number of projects are concerned with developing improved methods for the isolation and enumeration of salmonellae in eggs and egg products, meats, poultry, and freeze-dried foods. The results of these studies will contribute substantially to the body of available information concerning the prevalence of salmonellae in our environment and to the improvement of programs for the prevention and control of salmonellosis.

FOOD PROTECTION TRAINING

The training activities of the Division, as related to the control of salmonellosis, are centered on the presentation of specialized courses to members of industries that process and serve food, Federal agencies concerned with food protection, State and municipal health departments, and universities. These courses are designed to provide information on the epidemiological and laboratory investigation of food-borne disease outbreaks; and on procedures for the collection, examination, and evaluation of perishable foods to ensure safe handling in public eating establishments, aboard interstate carriers, in hospitals, in nursing homes, and in other institutions. The occurrence and sources of salmonellae in foods are discussed in these courses, and the "recycling phenomenon" is presented to emphasize the principle that food-borne salmonellosis is preventable, provided vigilance and interest are maintained and the links in the chain of sanitation barriers are preserved.

OTHER CONTRIBUTIONS

The Division conducts a number of ancillary activities that may, in fact, have a bearing on the prevention and control of salmonellosis. For instance, the Special Engineering Services Branch is concerned with sanitation in recreational areas. Undoubtedly the control of food services, water supplies, swimming areas, sewage disposal, and the like in public parks is important for the prevention of salmonellosis as well as other diseases.

This Branch also seeks to improve the disposal of solid wastes in urban areas. One of the methods being considered with the Tennessee Valley Authority is composting of refuse that has been macerated and mixed with raw sewage. The process is attractive from the economic standpoint and the residue may have agricultural uses, provided that salmonellae and other harmful agents can be destroyed during composting.

The Metropolitan Planning Branch deals with the environmental engineering problems arising from the growth of cities. It assists the suburban communities to foresee and provide for the sanitary facilities needed to cope with increased population density. Not only the extension of water and sewerage systems, but the timely adoption of food ordinances, codes and standards in consonance with the rest of the metropolitan area, are actions that will minimize the health hazards from enteric diseases, including salmonellosis.

CONCLUSION

Though much remains to be done, the combined activities of the various branches within the Division of Environmental Engineering and Food Protection have made substantial contributions to the recognition, prevention, and control of salmonellosis from foods and other environmental sources. The Division is in a favorable position to extend its research, technical assistance, and training functions, in coordination with other governmental agencies and industry, in order to achieve much needed improvements in protection of the public against salmonellosis. Our success will depend principally on the levels of support and cooperation that can be brought to bear on the problem.

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Panel Discussion

Dr. James H. Steele, Chairman,

Mr. Richard T. Carey, Dr. Charles F. Niven, Jr., Dr. Clarence H. Pals,

Dr. Glenn G. Slocum, Dr. John W. Walker, and Mr. Harold M. Williams

DR. STEELE: One thing I want to bring to your attention before starting with the questions, is the size of the problem we are dealing with. Accordingly, I should like to give you some statistics concerning food products of animal origin. Last year (1963), 109,000,000 animals were inspected by the Meat Inspection Division of the United States Department of Agriculture (USDA). These animals provided approximately 83 percent of all red meat produced in the United States—the equivalent of about 25,000,000,000 pounds. There are about 4,000,000,000 pounds of meat that come under some type of local inspection or are not inspected at all. About 1,300,000,000 pounds of red meat were imported from a total of approximately 35 different countries. The average consumption of red meat in the United States is about 170 pounds per person.

As to poultry products or what is referred to as white meat, last year, 2,700,000,000 broilers and fryers, about 300,000,000 hens, and about 100,000,000 turkeys were marketed, bringing the total of fowl to over 3,000,000,000. That totals approximately 7,000,000,000 pounds of meat or about 34 pounds per person. From this, you get some idea of the intake of poultry products versus that of red meat, the ratio being about one to five.

Eggs total about 8,000,000,000 pounds a year. Going on the basis that a dozen eggs weigh about 24 ounces, a person consumes a pound and one-half or about 350 eggs annually. A few years ago, that figure was a little higher, around 360 or 365 eggs annually.

The total amount of animal feed produced last year was about 180,000,000 tons, which may be a little low. That includes about 6,000,000,000 bushels of cereal grains consumed by animals.

These figures may give some idea of the magnitude of the problem that we are dealing with here.

DR. PALS: I would like to make one comment on the "83 percent—17 percent" figure that you gave to compare the amount of meat subject to Federal meat inspection with that covered by other forms of inspection coverage or no inspection. Last year about this time (March 1963) the Meat Inspection Division made a report to Congress concerning the present status of State meat inspection. Some of this 17 percent does receive adequate meat inspection at State or local levels. But when we realize that only 34 of our States have any laws covering meat inspection by the State, it leaves a rather large gap.

Some of the States have laws but very inadequate enforcement. President Johnson's 1964 Consumer Message called for adequate inspection of all meat sold in interstate or intrastate commerce. This is a goal which is going to be difficult to reach. However, President Kennedy made a similar appeal in his 1963 Consumer Message, and there has been considerable activity in the past year (1963) in States that do not have adequate programs. We are cooperating with those States in giving them all of the assistance that we can. We have not given them any money, but have provided advice and help in training their people. I think we have accomplished a considerable amount this past year.

Concerning our actual activities, the Meat Inspection Division has the responsibility of assuring that all meat and meat food products prepared under its supervision are clean, sound, wholesome, and fit for human consumption. To fulfill this responsibility, various procedures and requirements are enforced. First, everyday before operations commence, all facilities are given a thorough sanitary inspection. Any insanitary condition found would prevent the use of the area or equipment involved until the unsatisfactory condition was corrected.

At those establishments where slaughtering operations are conducted, all animals are examined prior to slaughter. Also, at the time of slaughter each carcass and organ are given a complete examination. These inspections are performed by graduate veterinarians and their trained assistants. All animals or tissues found to be diseased are condemned for human food purposes and destroyed under the supervision of an inspector of this Division.

All areas, equipment, and personnel used to handle the edible product must be sanitary. All equipment coming in contact with diseased or contaminated tissues are required to be cleaned and sterilized before being used again. The dressing procedure must be performed in such a manner as to reduce the possibility of contamination. For instance, the implements used to perform certain operations are required to be sterilized after each use. The hide must be removed in a manner that prevents contact between the external surface and the carcass. Also, the digestive tract is closed on both ends and removed intact to eliminate the possibility of contaminating the carcass with stomach or intestinal contents. The eviscerated carcass is then washed and promptly refrigerated.

The further handling of the carcass or parts and the production of meat food products are required to be conducted under the utmost sanitary conditions. The equipment used to prepare these products must be constructed to facilitate cleaning and dismantling for inspection. The nonmeat items used in preparing meat food products must all be approved before use.

The sanitation of all parts of the official establishment is supervised by Division employees. While the Meat Inspection Division is not responsible for controlling the preparation of feedstuff, we are confident our present sanitary practices have some beneficial effect. Also, if we were given the responsibility, we could exercise more control over inedible and condemned material.

> QUESTION: What is the effect of antibiotics in feed on the recovery of salmonellae from rectal swabs or excreta from animals eating this feed?

DR. NEWELL: This question came up in another panel discussion (page 101). I think that some evidence and some observations made on antibiotics in feed were not taken into account in the answer given to that panel.

Drs. Milner and Shaffer (1) have reported on the results of giving antibiotics to chicks artificially infected with various types of salmonellae.

They studied a variety of different antibiotics, and showed that giving these antibiotics in relatively small doses apparently does not affect the infection but does affect the excretion of salmonellae, at least during the period when the antibiotics are being given. When the antibiotics are stopped, excretion very frequently starts again. In addition to this work in chicks, some of the work of Dr. Paul Williams (2) on pigs in Louisiana has pointed out the possibility that this similar series of events can happen in pigs. The excretion of salmonellae by pigs fed antibiotic feeds is low; in fact, salmonellae may not be detected at all. However, when the antibiotics are stopped, even if no further salmonella infect the animal, excretion appears to start again. In other words, the antibiotics apparently have not affected the infection rate of these animals but have affected the excretion while the antibiotics have been given.

Considering the widespread use of feeds containing antibiotics in this country, it seems highly probable that in some of the investigations on farms, even those involving S. derby, the low recovery rate or the lack of any recoveries of salmonellae may be related to the use of these feeds.

This also raises the possibility that the apparent build-up of salmonella excretion by animals from farms through the holding pens to the slaughter house, can possibly be explained by the same reasoning. On the farms, there is apparently a very low rate of excretion which may be kept low because of the antibiotic feeding. On the way from the farms through the holding pens to the slaughter houses, antibiotics are stopped; and this is where a build-up apparently occurs. Maybe, this build-up is not a cross infection from animal to animal. It may be just an expected effect from the stopping of antibiotics in the feed.

> QUESTION: Are the problems of control of salmonellosis in eggs so different in three countries — the United States, England, and Canada — that legislation or national measures are needed in one and not in the other?

MR. CAREY: In this question, you quite obviously want to know about pasteurization or compulsory pasteurization. In Canada, the law says that you cannot transport, hold, or possess eggs that contain salmonella. In England, the law defines how eggs are to be pasteurized and what tests are to be used to check on the effectiveness of pasteurization. In the United States, our feeling right now is, and this is in agreement with Mr. Williams, that the problem might best be solved by industry taking care of it rather than the USDA going in and saying "eggs have to be pasteurized." If voluntary action does not resolve the problem, then I am quite sure compulsory measures will have to be taken on a national level.

DR. NEWELL: Is it agreed by the panel that pasteurization is necessary in this country?

MR. CAREY: I do not know that it is necessary right now. Frankly, I would rather see the problem controlled by something other than pasteurization. If the salmonellosis problem continues to grow, I think the egg industry is going to be hurt to such an extent that they will have to do the pasteurization on their own accord. This will not solve the problem of shell eggs going to the housewife.

I think we can correct this problem by pasteurization but I still feel that the best answer to the problem is to correct it at its source. This should theoretically take care of shell, frozen, and dried eggs.

One important point is that the pasteurization of eggs must be efficient. If you can assume a 99 percent level of efficiency of pasteurization and if the original level of salmonella is quite low, the incidence or chance of recovery of salmonella in the finished product is eliminated or else reduced to what would be an insignificant level.

DR. THATCHER: I speak because Canadian regulations were mentioned. I would like to describe how it was that the Food and Drugs Directorate of Canada reached the decision to introduce a regulation which states that "no person shall sell any egg product for use as food unless it is free from organisms of the genus salmonella, as determined by the official method." This is not a unilateral development; it followed our normal procedure which is to discuss all aspects of a problem with representatives of the industries that are directly involved or might be involved.

The poultry and egg industries, through their associations, were invited to the offices of the Food and Drug Directorate in Ottawa, and the entire story as we knew it was aired with them. Similarly, representatives of the industries concerned with the manufacture of cake mixes also were invited to Ottawa. An overwhelming majority of these delegates endorsed the proposal which we had offered to them in draft form and which eventually emerged as I have just cited. This was a corporate development. As to the question of whether pasteurization of egg products should be mandatory or not, I think this question involves a principle that we ought to consider rather carefully. The principle I believe in and the one which has become a policy in my Directorate is that the choice of a technological procedure and freedom to develop new processing procedures are the prerogatives of industry. It is however, the responsibility both of industry and of health authorities to see that whatever technology is used, is effective. It is a responsibility of my organization to minimize disease risk to the community. The evidence that disease is occurring is positive. Therefore, the necessary control measures must be applied. Our concern is not how salmonella are removed from eggs, but that they are! This point of view is shared with us by the two Canadian industries I have referred to.

DR. COCKBURN: It would be a great mistake not to have clearly in our minds that this problem can be divided into three distinct parts. The first is the duck egg, the second is the hen egg, and the third is the egg product There can be no doubt that the duck egg must be treated in some special way if it is going to be used on a large scale. It must be pasteurized because you cannot guarantee the freedom of the duck egg from salmonella. The whole of the literature and the whole of practical experience supports this view.

As far as the hen egg in the shell is concerned, the situation is entirely different. The shell egg is a pretty safe product, and I do not think one should consider special methods beyond hygienic production methods for dealing with it.

Concerning egg products, in the United Kingdom careful observations have been made on hygienic egg processing plants over a period of years. It has been seen that you can reduce or eliminate the amount of contamination for certain periods. But occasionally, contamination recurs, and you do not know how long it is going to last. Thus, with the egg product, whatever its origin, you have to end up with some form of heat treatment. That does not mean you should not produce your eggs hygienically. You cannot produce "dirty milk" and then pasteurize it and be satisfied that you have a good product. It is exactly the same thing with egg products. They have to be clean to begin with and have to be pasteurized before they are distributed.

DR. HOBBS: In England, the industry cooperates wholeheartedly with us in the effort to find the right conditions for making all egg products safe. They were tired of criticism and of knowing that the food product with which they were intimately concerned was causing illness, often of as serious a nature as paratyphoid fever. Much money has been lost due to contaminated material. Machinery and establishments were provided for experiments and many producers were extremely cooperative and helpful; without this help, it would have been difficult to find practicable times and temperatures of treatment. The industry is relieved now that the regulations are in force. I did not want you to think that they had been forced to accept the measures. The manufacturers and the users wanted the regulations, and the Egg Marketing Board through Dr. Knowles has been working with us towards this end.

I would say that at the present time the only practicable way to be sure that egg products are safe for human consumption is by pasteurization, using a sufficiently high temperature for a short period of time.

QUESTION: You stated it would be a long time before the salmonella problem would be solved. Why do you think it will take so long when so much is already known about the problem?

DR. NIVEN: I think the question pretty well answers itself. Because we know so much about it, we know it will take a long time. We must keep in mind the many and varied aspects of the problem. One example might be the fact that the same serotype can infect many different species of animals and also man. Each can become a carrier. Realizing this, how can we expect to eradicate salmonella from the face of this globe? I think we are going to have to contend with salmonellae for a long time but yet learn to live with them. Additionally, the fact that salmonellosis is not generally a highly acute and fatal disease makes it more difficult to control than a more virulent disease.

I would like to go out on the limb on one thing. Please do not interpret this as a defensive statement at all by me. If we fed every animal in this country sterile feeds, I am sure we would continue to have a salmonella problem among our livestock as well as humans, although the problem might be lessened.

QUESTION: What is the average age of an egg when purchased by a consumer?

MR. HAROLD WILLIAMS: There are 180 or 190 million consumers scattered throughout the country and the system of distribution varies in each area. I would say anywhere from about 4 days to 2 weeks.

QUESTION: When will the consumer be able to buy fresh eggs on the market that are not more than 48 to 72 hours old?

MR. HAROLD WILLIAMS: There is a current trend to establish large production units of hens around the cities. I know for instance that around Chicago, there is a large operator who has well over 100,000 hens. He has an arrangement with a company that delivers juice to homes, and they pick up and deliver the eggs daily. I do not imagine those eggs would be over 48 to 72 hours old. There are also some other units going up around Chicago.

QUESTION: At this time, the distribution of cracked eggs appears to be dangerous and yet of only marginal importance to the poultry industry. If the poultry industry intends, in fact, to cooperate, why do they not require or recommend that all such eggs be sent for "breaking and pasteurization"?

MR. HAROLD WILLIAMS: I think that might be a good idea.

DR. STEELE: There is one question along this line I would like to have your opinion on, Mr. Williams. One of the State health officers was discussing my comments that grade A eggs for institutions and hospitals are quite safe. But, I went beyond that. I said that if a hospital really wants to protect themselves, why do they not contract with some of the new suppliers coming into existence so that they would have daily deliveries of fresh eggs from one source, and they would practically have a guarantee that the eggs are being produced from a disease-free flock under the most hygienic conditions possible. What would you think about that for a recommendation to the hospital purchasers?

MR. HAROLD WILLIAMS: I think it would be an excellent idea.

DR. STEELE: Would a hospital administrator like to make a comment on what the hospital's point of view on this would be?

DR. HOOK: I can give you a comment on it. Hospitals are very unenthusiastic about dealing with a single dealer from the standpoint of competitive prices. They always attempt to deal with more than one group. I do not know that this is a valid point, but it is a point that they make.

DR. STEELE: On the other hand, it seems to me they would be buying additional protection, and this is something you would pay for under these circumstances.

QUESTION: If the interstate transporting of salmonella contaminated foods is controlled by the Food and Drug Administration, how does Dr. Slocum explain the interstate passage of grossly contaminated egg products as described by Dr. Ager in Washington?

DR. SLOCUM: Approximately eight billion pounds of egg products are currently being produced per year. I do not know about the number of egg-breaking establishments in the United States. Obviously to cover all of this tremendous industry at once is an impossibility. The industry is quite aware of the position of the Food and Drug Administration.

I think it is obvious that we must rely a great deal upon a well intentioned and well informed industry to do the bulk of the job of eliminating salmonella from these products. We do have a two-phase program in operation. The first part is to find what we call hazardous uses of egg products, that is, instances in which raw egg products are added to prepared foods which receive no further heating before consumption by the consumer. Such situations obviously will represent the greatest health hazard from the egg products as contrasted to their use in foods which are thoroughly cooked. We are implementing this approach as fast as possible in all our 18 districts. We are scheduling inspections as rapidly as this can be accomplished, but it is going to take a long time to cover the wide variety of uses of eggs. We are hoping the program will call the users' attention to the hazards of accepting infected products.

The other aspect of the program is the inspection of the egg-breaking establishments, and we hope by this means to find the conditions which result in gross contamination of the products. Certainly, this will again call the attention of the individual producer to the fact that we do regard these products as adulterated and that it is essential to eliminate contamination. Sometimes a little control goes a long way.

> QUESTION: When dressed fowl sold in the market are found contaminated with salmonella, are they considered unsafe and subject to confiscation?

DR. SLOCUM: I think this is two separate questions. As to whether they are unsafe, certainly poultry products have been implicated perhaps more than any other single food group in outbreaks of salmonellosis. Salmonella contaminated fowl that are undercooked or are recontaminated after cooking are unsafe.

As far as control or confiscation is concerned, the basic control of wholesomeness is carried out under the Poultry Products Inspection Act. The Food and Drug Administration does not participate in the factory inspections or in the detailed control of the Act. The Act can be used to confiscate known dangerous products; but, in practice, there is very close and excellent cooperation between the Food and Drug Administration, the Meat Inspection Division, and the Poultry Products Inspection Division. If one or the other of these groups hears of an outbreak, then the samples obtained are shared. The confiscation of such goods is only invoked where it is essential to remove a product from the market that cannot be removed by another method.

DR. NEWELL: Are the regulations that exist forbidding interstate passage of salmonella contaminated foods unenforceable; and, therefore, should they be neglected in our consideration of the control of this problem?

DR. SLOCUM: I think these regulations should not be neglected and are an extremely potent force. They are enforceable as applied to specific shipments or lots of egg products moving in interstate commerce, but it is not the individual action that is ultimately desired. There is a great deal of protection in removing contaminated products from the market, but it is the effect that this brings about in production that is most important.

We do not require specific treatment processes any more than Canada does or the State of Washington. We rely upon the adulteration provisions of the law which says a product is adulterated if it contains a poisonous or deleterious substance. Enforcement action taken by the Food and Drug Administration brings about a chain reaction, which leads to quite rapid correction of many of these problems.

DR. NEWELL: If it is true that this law does take effect in a slow but regular way, the law has been present for some considerable time, and yet it has been brought out by quite a number of people in this conference, that many of these egg products are contaminated and possibly have been contaminated for a considerable time. If one is to judge this law by its effectiveness, its value seems to be minimal.

DR. SLOCUM: I think Dr. Newell certainly is correct. There has been, I think, a long delay in the application of this law to the problem. When we became aware of the fact of contamination, largely as a result of the English investigations in 1945, the question arose in this country, "Why are we not seeing outbreaks traceable to eggs?"

We all have gone over many of the reasons for this, including a lack of adequate investigation and reporting. But the fact remains that there were very few instances reported, and it took us many years before we found clearly documented cases of salmonella infections traceable to egg products. The fact that this is the first national meeting of this sort that I have ever attended or heard about indicates that this problem is coming into real focus only now, although we have known for a long time that contamination existed.

The very fact that salmonella contamination in egg products is widespread and that these organisms are commonly present in poultry is probably the problem. It is a serious action to initiate a program which is going to change the whole production and whole economy of the industry. We worked too slowly in initiating corrective action, but we have taken action in connection with specific outbreaks. Now we are applying control procedures across the board on all egg products.

MR. HENNINGSEN: I am representing my industry as Chairman of the Egg Processing Committee of the Institute of American Poultry Industries, whose membership is made up of all of the major driers and most of the major frozen egg processors in the United States.

I believe there are certain facts about egg processing that may prove of value to the participants in this conference and perhaps give a clearer view of what the egg processors are doing about control of salmonella.

Much discussion has been devoted to the role of pasteurization in the control of salmonella. Certain members of the egg processing industry began pasteurization of egg products in 1937. Routine identification of salmonella and determination of its presence in egg products began in 1944. I wish I could say that all egg products are pasteurized, all egg products are checked for salmonella, and all egg products are salmonella negative. I very obviously cannot, but I can tell you that of the 21 egg driers operating in the United States, all have pasteurization equipment, and all are using this equipment. Of the major users of dried egg products, I know only one company that does not have a rigid salmonella control in their product specifications.

The frozen egg industry, you must understand, is larger than the dried egg industry not only in volume, but in number of participants. Processors range from large organizations conducting multiplant operations to small family proprietorships, and only about 50 percent are equipped to pasteurize. At this time, however, many are making the investment to properly equip themselves. It is either that, or get out of the business. Users of frozen eggs, as well as the users of dried products, are rapidly writing salmonella controls into their product specifications.

I would like to point out that we in the egg processing industry do not look at pasteurization as the complete solution to salmonella control. For one thing, we find different people have different concepts of what pasteurizing means. The classical definition, "to heat for a period of time sufficient to kill all harmful bacteria," does not fit eggs. The egg processor does not and cannot accept this classic procedure, as he would heat damage his product the point of denaturation in attempting to make it sterile. Rather, he considers pasteurization to be effective if total count is under 25,000 organisms per gm. or ml., if the coliform count is under 10 organisms per gm. or ml., and the product is salmonella negative.

There is a lot we egg processors have to learn about pasteurizing this very complex colloidal protein called egg, but I can assure you we will not learn about it if we are forced by regulation to pasteurize all products at a set time and temperature. Should this happen, I believe work currently going on to improve egg processing methods to control salmonella and other bacterial contaminants will be seriously affected. Rather, we would hope that our government would take an attitude similar to that of Canada. Dr. Thatcher has outlined very clearly that in Canada the government advises industry what standards the finished product must conform to and then the government allows the processor/manufacturer to work it out in his own way. Believe me, the people in our industry are very well aware of the problem of salmonella and are doing something about it.

When you move a procedure out of the research laboratory into production, you must adopt a standard procedure that becomes routine. You cannot be forever changing procedures in a production situation. This holds particularly true for determining the presence of salmonella. Industry needs your cooperation in arriving at a uniform salmonella determination method, a uniform sample size, and a uniform sterile sampling technique. Unless we have these, the industry is going to be in for a great deal of unnecessary trouble.

For all practical purposes, we have a standard test. Since the Food and Drug Administration is the enforcement agency and the North Method (3) is their choice, it automatically becomes the standard by which industry operates.

Finally, we ask your consideration in working with us through what may prove to be a difficult time. We believe as you do that salmonella can be eliminated; however, it must be a cooperative effort, and not a punitive one, where this industry or that industry is held up as the horrible example. We are taking the steps to put our house in order. We will depend on and welcome your help and guidance.

DR. STEELE: Thank you, Mr. Henningsen, for your comments.

> QUESTION: What cooperation do you think industry can receive from the transportation companies?

DR. WALKER: Initially, in dealing with common carriers such as railroad people and truckers, it was necessary to explain the possibility of animal and poultry feed ingredients becoming contaminated in transit. It has been demonstrated that contamination of carriers may contribute to the maintenance of the salmonella cycle. Now that the carriers understand the problem, they want to try to do something about it.

> QUESTION: When do you feel the majority of the rendering plants will be able to initiate effective control of salmonella contamination and delivery of salmonella-free products?

DR. WALKER: At the present, we do not know that the majority of the plants are not selling salmonella-free products. In working on this, we have discussed the situation with representatives of the rendering industry and found that approximately six large organizations produce 80 percent or more of the rendered protein material manufactured in this country. Thus, if we can first make certain that these companies sell a product that is free of salmonella, we will have 80 percent of the potential problem corrected. However, you have to consider that the remaining 20 percent is made up of hundreds of small operations which may involve only one or two cookers. Here may lie the heart of the problem, and effecting a cleanup in these plants will take some time. I feel certain that the rendering and fish industries are doing a great deal in their own right in trying to make certain that their product does not get recontaminated with salmonella.

QUESTION: Are we to assume with reasonable assurance that pig pens, poultry farms, and sausage kitchens will become free of salmonella if feed companies will produce salmonella-free products?

DR. WALKER: I feel it would be expecting too much to say that salmonella problems in the places named will become free of salmonella if feed companies produce only salmonella-free products. Certainly it will contribute its share to helping the over-all situation.

QUESTION: Would salmonella be perpetuated in chicken farms and pig pens even though feed is salmonella free?

DR. WALKER: Chicken farms are not the same operations as they were 10 years ago. Chicken breeding and growing operations have become very big business with fewer poultry men than there were 10 years ago. Approximately 10 primary breeders in the country produce 90 percent of the multiplier chickens for broiler production. The number of premises and operations involved, especially the smaller farms, are fewer in number each year. This makes the job of stopping the perpetuation of salmonella on farms more reasonable and attainable. I think eventually the salmonella cycle in this area can be broken.

QUESTION: The domestic animal salmonella cycle must be broken in order to reasonably influence human disease. It is difficult to accept the continued distribution of salmonella-infected feeding stuffs. Why should not the first step be a requirement that noninfected feeds be clearly labeled as such?

DR. WALKER: In the United States Department of Agriculture's Recommended Sanitation Guidelines for Processors of Poultry and Animal By-Products, (4) which we developed in cooperation with the industries concerned, this point is brought out. If feed ingredients which receive special treatment or care in their preparation can be so marked or recognized, this would serve to stimulate their use by the feed industry. The idea of labeling salmonella-free products could also be used by the feed industry; but we must remember that as of now, there is not a practical, surefire way to treat contaminated feed on a large scale to make it free of salmonella. We know it is being done in a small way in specialty feeds, but this is something different and expensive. Prevention of contamination is something attainable and practical. This is what we hope to do in applying the sanitation guidelines.

DR. DARBY: I agree wholeheartedly with Dr. Walker but want to caution everyone that you are dealing with an industry that is producing 40 million tons of commercial feeds annually, and then there are probably 40 million additional tons produced on farms. If you were in a position to produce a practical, salmonella-free feed, the chances of recontamination are very great. I think the animal feed industry, as Dr. Rossow has indicated, is going to do the best possible practical job in producing feeds that are free of contamination.

QUESTION: What is the difference between expansion extruded and pelleting extruded feeds?

DR. DARBY: This is the technical terminology that we obtained from our engineering department. In the pelleting process, the meal is subjected to two phases; first, the meal is conditioned by injecting steam into it until the moisture level is between 11 and 19 percent. The next phase is to force the moist meal through a dye which then results in a product that is firm, dense, and in the form of a pellet. During this entire process the meal is exposed to temperatures of 160° to 180° F. (71° to 80° C.). The length of the process will vary between 2 to 16 seconds depending upon the speed of the operation. The important factors of sterilization or pasteurization are time, temperature, pressure, and moisture. With these variables, we can readily understand why there is a difference of opinion concerning the effectiveness of pelleting in eliminating salmonella organisms from feeds.

The expansion extrusion refers to a recently developed process that conditions the meal with steam until levels of 25 to 35 percent moisture are attained. The final step renders the product into a form that is the opposite of pelleting. Instead of concentrating the meal as it does in pelleting, it causes the expansion of the meal to a point where the product has a greater surface area and is of a lesser density. To assist in this pasteurization process, temperatures of 200° to 350° F. (93° to 176° C.) are reached for 45 to 60 seconds. Under these conditions, we have never been able to isolate salmonella organisms from the processed feeds.

MR. TURNBULL: Poultry industry members have asked me to state that it was their hope that conferees would not get the idea from comments made earlier that the poultry industry does not recognize the presence of a problem.

We recognize that our products are frequently involved in outbreaks of food poisoning, and we recognize our obligation and that of the public health authorities to prevent such occurrences wherever possible. We expect to work with them.

We do ask, however, that all public health officials in turn recognize the need for carefully worded statements to the press and the public. Improper phraseeology can cause needless financial losses to industry members and serve no purpose with respect to solving the problem of salmonellosis.

For example, the use of the phrase 'hospital-associated' in the news releases in June and July of 1963 could have carried two connotations. In fact, I made a long distance call to the Poultry and Egg National Board in Chicago to inquire if that agency had determined whether this meant the infection had become so severe that attending physicians felt the victims should be hospitalized or whether it meant the infection had been picked up by patients already confined in a hospital.

Further, more emphasis should be placed on proper food handling rather than on product defamation. For example, an infected food handler may be the source of infection, regardless of whether the food served is eggs, fish, poultry, or gravy. However, the news stories usually place the blame on the food itself rather than on improper preparation. The public needs to be educated to the fact that proper food handling can reduce threats to health. A good example is the manner in which the public has been educated to cook all pork thoroughly and thus prevent trichinosis.

QUESTION: What developmental steps must be taken before radiation techniques may be used on a commercial scale for the control of bacterial infections in food and food products?

DR. DARBY: Many years ago, we ran tests with feed stuffs contaminated with mixed bacterial cultures. These were sent to the University of Michigan for irradiation; and, as Dr. Rossow indicated, it was demonstrated that irradiation did sterilize these feed products. It is our impression that, at the present time, the process is too costly; and we also believe there are some adverse effects of irradiation on some of the ingredients. So from a feeding standpoint, we felt that something is left to be desired. But I want to hasten to add that I do not feel that I am qualified as an expert in this field.

We do know that there is one experimental product available on a semicommercial basis for specialized uses where the pasteurization process has been by irradiation.

DR. SHEA: The cost of irradiation would be about 1/2 to 1-1/2 cents a pound depending upon the product and particular application involved. There are other applications where the use of irradiation to destroy salmonella or other parasites, in order to extend the shelf life of a valuable food item, is not as expensive.

DR. DARBY: Is this being used at the present time for feed stuffs?

DR. SHEA: No.

DR. EL-BISI: I should like to comment with regard to the application of radiation energy for the preservation of foods and feeds. The Food Division of the U. S. Army Natick Laboratories, Natick, Massachusetts, have been heavily engaged over the past 10 to 15 years in a Research and Development program geared towards the development of radio-canned foods, namely foods which are packed in tin cans or flexible-film packages, hermetically sealed and irradiated at high-dose levels (3-1/2 to 5-1/2 Mrads). The primary objective in this program has been to provide the U. S. Armed Forces with a potential source of stable, wholesome, and morale-boosting foods analogous in shelf-life to thermo-canned foods. The Atomic Energy Commission (AEC) has been simultaneously involved in Research and Development activities geared towards the civilian development of low-dose radiation for the pasteurization of perishable fruits, vegetables, and meats to lengthen refrigerated shelf-life. Unlike the AEC, most of our Research and Development activities at Natick have been an in-house effort, coordinated with several outside contract activities.

Our concern about the microbiological safety and stability of the radio-canned products has been focused upon the spores of Clostridium botulinum, since it is the most resistant spore population tested. Our studies on Cl. botulinum spores have encompassed cellular, environmental, and process variables which would influence or govern the radioresistance of these spores. We are currently planning fundamental research on the botulinus toxin and its radioresistance. We are also attempting to coordinate efforts with the Robert A. Taft Center to study the incidence and radioresistance of enteric viruses in foods to be irradiated.

Along with these microbiological studies, our colleagues in the Irradiated Food and Radiation Source Branches have been concerned primarily with the development of new irradiated products as well as the development of the radiation sources and delineating dosimetry problems.

Currently, we have a unique radiation source facility which is comprised of a linear accelerator of 24 MeV with a dose rate of up to the 10th rads per second, as well as 1.3 mega curie gamma cell with a dose rate of about 4 Mrads per hour.

Throughout our broad fundamental studies, we have been confronted with various questions and problems related to a wide variety of food items; and, hence, our staff would be capable of advising on the applicability of irradiation energy to food or feed systems with regard to the salmonella problem and would welcome and encourage any inquiries along these lines.

> QUESTION: Should all food poisoning salmonellae be attacked, that is salmonella as a whole, or should selective control be undertaken for those serotypes most important to man?

DR. STEELE: Epidemiologically, there is nothing that will distinguish between the various salmonellae. Therefore, all salmonella should be attacked in order to reduce the total challenge to human health.

> QUESTION: Which would be most reasonable (a) to decrease the amount of human illness and mortality by attempting to protect the most susceptible humans (b) or by eliminating or controlling the presence of such ubiquitous agents?

DR. STEELE: I think it is quite obvious that this problem should be undertaken as a whole problem. It is going to affect animal health as well as human health and to separate them would be foolish. Therefore, we must work to eliminate the threat of these ubiquitous agents.

My last comment is in regard to control procedures themselves and the research necessary. When we consider that the human food industry in the United States today is approximately a 75 billion dollar industry and that the animal food industry is just about one-half of that — 35 billion — we have a total exceeding 100 billion, which is more than one-sixth of our gross national product. It seems very logical that industry, government, and anyone else concerned must give more consideration to the appropriations that will be going into this area in the future. What will be the amount of food, the number of animals, the number of human beings by the end of the century? The salmonella problem will be compounded if it is not tackled today. And today is almost too late to begin, as so many of the participants of our audience and panel have pointed out.

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BANQUET SPEECH

Dr. W. Charles Cockburn

Banquet Speech -

Salmonella, Retrospect and Prospect

W. Charles Cockburn, M.D., M.R.C.P., D.P.H.[1]

The program prepared by Dr. Goddard and his committee for this first National Conference on Salmonellosis covers almost all aspects of the subject; and, in order to keep off the preserves of others, I decided to look at some parts of the history of the disease in the belief that they are of interest and still provide good and apposite illustrations of the complexity of the subject. Mr. Churchill is reputed to have said that the further you can look back the further you can look forward. After recalling some early observations, I should like to look a little way to the future, leaving most of the present in the capable hands of the conference speakers.

THE PAST

Bacteriological knowledge of salmonellosis began in 1888 when Gaertner isolated the Gaertner bacillus. But about a decade earlier Bollinger made a significant contribution to the epidemiology of bacterial food poisoning by his study in Germany of human illness associated with meat from sick or parturient animals. The reports of Bollinger and later of Ostertag showed very clearly the importance in those days of meat from sick animals, in outbreaks of food poisoning. In one series of 48 outbreaks, 14 animals had gastroenteritis, 11 were parturient or immediately post-parturient; 13 had mastitis or other forms of sepsis; 8 were moribund, and only 2 animals were neither sick or parturient. Meat from sick animals is seldom incriminated now in the affluent countries, but it is still eaten eagerly in countries where meat is one of the scarcest of scarce foods. When the health services in these countries become adequate, we may expect to have more reports of these outbreaks associated with food of this kind. There are already quite a number of reports of outbreaks associated with meat from sick camels, and it is odd to find that most of the strains of S. typhi-murium isolated in these outbreaks are anaerogenic.

Many of the early outbreaks were thoroughly investigated, and most of Bollinger's conclusions have stood the test of time. One observation, however, which has received little attention from later investigators would not only lend itself to controlled studies, but would undoubtedly attract volunteers. Bollinger mentions two outbreaks in which drinking alcohol had a protective effect. In one, a girl who drank brandy along with the incriminated beef was not ill; and, in the second, it is said that those who drank plenty of alcohol were less ill than the others!

This early work is well known. Rather less well known are the early reports from England. In 1880, Ballard investigated the first fully reported outbreak of what was probably food-borne salmonellosis (1). The title of his paper was lengthy – "An acute specific disease characterized by a peculiar diarrhoea epidemic among persons who had partaken of refreshments provided at a sale on the Duke of Portland's Estate at Welbeck."

Ballard made a thorough investigation. He collected information on those who had eaten the suspected food at the sale and also about those who had not attended the sale but had eaten food carried home by friends or relatives. He noted that the attack rates were highest in those who ate the food 2 to 3 days after it had been cooked and lowest in those who ate it 1 day after it was cooked, and that the incubation period was between 12 and 48 hours – usually 12-36 hours. He made other useful observations which later generations of epidemiologists have often repeated. In this outbreak, at least 72 cases occurred, 4 of which were fatal. The food responsible was salted ham which had been soaked to remove the salt then smoked and finally cooked, skinned, allowed to cool and sliced. An American author describing Ballard's report some years ago said that the hams came from "abroad."

They came in fact from the United States of America, but the infection was probably introduced during some of the numerous processes to which the hams were subjected after arrival in England. Klein fed portions of ham to dogs, cats, rabbits,

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guinea pigs, and mice; he isolated in cultures of egg albumen, an organism which he called the "Welbeck Bacillus." This was probably the causal agent and was probably a salmonella. Ballard's experience at Welbeck awakened his interest in episodes of this kind, and he collected first hand or from the literature information about 14 outbreaks which occurred in England between 1878 and 1890 (2). He developed the very useful habit of recording the intervals from ingestion of the food to the onset of illness – information seldom given for the early outbreaks reported from the continent of Europe. One of the outbreaks reported was associated with American canned pigs' tongues and occurred in 1882. As the incubation period was 4 hours, this may well have been one of the earliest staphylococcal outbreaks to be reported – though, of course, there was no suggestion then that staphylococci were responsible.

Comparison of the early continental and English outbreaks brings clearly to light the differences in the epidemiology of food poisoning in different countries and cultures. The continental outbreaks were usually associated with raw or freshly cooked meat or sausages; whereas the English outbreaks were invariably associated with processed meats, such as hams and canned foods, or made-up meats such as brawn and meat pies. This distinction still holds good, although to a lesser extent than formerly.

In countries where they are widely used, eggs and egg products are one of the most frequent vehicles for the distribution of salmonellae.

Eggs, especially duck eggs, were suspected of causing trouble only a little later than meat and meat products. Two doctoral theses in the library of the University of Bordeaux, one by J. Peytoureau (1902) and one by André Le Coq (1906) are important sources of early information. Peytoureau's thesis was entitled "A study on poisonings by cream pastries" and Le Coq's "A contribution to the study of poisonings by cream cakes."

Peytoureau listed a number of outbreaks associated with cream cakes and similar items of food. In the first in Paris in 1829, a woman prepared a cream cake for her blacksmith husband who was stricken with colic and died about 3 weeks later. His wife was accused of poisoning him and was sentenced to hard labour for life. Between 1850 and 1901, other outbreaks were reported from Paris, Altona and Berlin (Germany), Algeria, Sheffield and Lambeth (England), and various parts of France. In 1902, more outbreaks than usual occurred in France. One, a large one, was associated with the Feast of St. Peter in Bordeaux, where 150 became ill about 24 hours after eating cream cakes. Peytoureau investigated a number of these outbreaks and studied the records of others. He noted that the interval from ingestion of the food until the onset of symptoms was usually between 12-48 hours but was sometimes between 4 and 8 hours. Outbreaks occurred at all seasons, but were specially common on feast days.

He considered the separate constituents of the creams as possible causes of the poisonings and was satisfied that none of the ingredients were suspect except vanilla essence and eggs, both of which had been used in all the creams. He could not decide which of the two were at fault. He pointed out, however, that eggs preserved in summer and used in winter often caused illness; that tanners, glovemakers, dyers, photographers, and others used the albumen of the preserved eggs and sold the yolks to the confectioners and biscuit makers "dirt cheap"; that both albumen and yolks had caused illness at one time or another as was shown by the records of outbreaks in England and Germany. In the Bordeaux outbreak of 1902 in which he was specially interested, preserved duck eggs had been used. Colleagues of his had in the past observed that persons who ate cream containing duck eggs sometimes became ill. Ducks were dirty feeders and infection might perhaps spread from the intestine to the egg without causing illness in the duck. Though duck eggs were bought freely by the inhabitants of Bordeaux itself, they were rejected with superstitious terror by the peasants. Peytoureau's suspicions of the duck eggs were stronger than his suspicions of the vanilla essence.

Four years later Le Coq reinvestigated the problem. Outbreaks associated with cream cakes which usually contained cream made with egg white had continued to increase in France. Le Coq obtained records of about 35 separate outbreaks. Not all were associated with cream cakes; some were associated with "cream snow" a mixture of egg white, sugar, and cream. The most dangerous cream was one made of egg yolk, gelatin, sugar, flour, and milk brought to the boil and then folded into raw beaten-up egg white. Hen eggs in the shell and dried albumen and egg yolks (to some of which boric acid was added as a preservative) were used in addition to duck eggs.

Le Coq described, as did Peytoureau, outbreaks with both short and long incubation periods, but apparently had no idea that they might be due to different etiological agents. After weighing the evidence for and against the different ingredients, Le Coq came firmly to the conclusion that the egg was the most likely source of the contamination, and that duck eggs and their products were more dangerous than hen eggs. The duck egg was the greater risk because ducks ate their food and laid their eggs in places liable to bacterial contamination and because there was evidence that foreign bodies, straws, seeds, stones, etc., were sometimes found in duck eggs having been pushed into the oviduct during copulation. If foreign bodies could reach the oviduct in this way, no doubt bacterial contaminants could do the same. Prevention, said Le Coq, could only be attained by ensuring that creams were not sold unless the egg in them had been heat treated.

The two theses were published some 14 to 18 years after Gaertner first isolated S. enteritidis. Le Coq speaks of possible bacterial contaminants but apparently he made no attempt to confirm his epidemiological observations by bacteriological examinations. The reports, buried in the library of the University of Bordeaux, had little or no effect on subsequent work on salmonellae in eggs.

In the United Kingdom, there is said to have been an outbreak associated with duck egg albumen in 1913; this was never published, and W. M. Scott was the first to become interested in the subject. He reported in 1926 a fatal case due to S. typhimurium in a child who ate a duck egg. In the following year, he studied three outbreaks in one of which 300 cases occurred in persons who ate ice cream. About this time, numerous reports began to appear from Germany, and many experimental studies were set up, both in Germany and the United Kingdom, which shed light on the various methods by which duck eggs could become infected. Because of these studies, Sieke (3) in Germany and Scott (4) in England suggested all duck eggs should be boiled for 8 minutes. Mainly because of the human desire to "add another minute or so for safety's sake" Clarenburg and Burger (5) added 2 minutes and Kraneveld and Mansjoer (6) recommended one more — making 11. After another few years, the British advocated 15. By this time, the eggs were probably 100 percent safe, but they were also stone hard. Fried eggs were also investigated. Kraneveld and Mansjoer fried them on one side for 5 minutes, but found about 10 percent still yielded salmonellae. After being fried for 4 to 5 minutes on both sides, they were sterile but practically uneatable.

Egg products were unsuspected in the United Kingdom until 1935 though ever since about the First World War millions of hundredweights had been imported annually. We owe the first report to Anderson et al. (7). In Wales, they investigated three outbreaks of infection with S. thompson associated with cream confectionery from one bakery in which frozen egg white from Shanghai had been used about the time the contaminated cakes were prepared. None of the egg white remained for examination so the epidemiological evidence was not bacteriologically confirmed.

Curiously enough when American spray-dried egg began to be imported during the Second World War, it was quickly suspected and examined (8). About 8 percent of samples were found to be contaminated. One cannot be certain why this vehicle of infection was so rapidly traced, but it should be noted that for the first time in Britain, a national Public Health Laboratory Service was functioning and had a system for the centralized collection of epidemiological information and the provision of reference services for the identification of unusual serotypes of salmonella. This meant that the appearance of exogenous strains of salmonella was quickly detected and the health services alerted. To this period, the United Kingdom owes the introduction of many strains which are now old acquaintances there: S. anatum, S. give, S. kentucky, S. montevideo, S. oranienburg, and S. tennessee. At the time, protein foods were scarce, and it was decided to continue imports and to attempt to reduce the risks by advising users not to leave the egg uncooked for long periods after it was reconstituted, and as far as possible to cook it thoroughly.

The experience with dried egg might reasonably have been expected to stimulate the examination of egg products from other countries, but this did not happen for a long time. It was Dr. Kenneth Newell's (formerly of Central Public Health Laboratory, London, England) dogged search in bakeries and confectioners' shops in Middlesex during an outbreak of paratyphoid fever in 1955 that led to the discovery that Chinese egg products contained salmonellae of many types including S. paratyphi B. Up to 19 percent of samples from some consignments of frozen egg or dried egg albumen yielded salmonellae, and nearly all the types isolated were those which had been responsible for most of the human cases as far back as records could show. Though S. paratyphi B. was only isolated from Chinese egg products, many other types of salmonellae were found in the products of other countries.

The so-called rodent viruses present another odd chapter in the history of salmonellosis.

About 1892, as a means of destroying field mice, Loeffler used cultures of the S. typhi-murium bacillus which he had isolated during a natural outbreak in white mice. A little later other workers used suspensions of intestinal pathogens isolated from ground squirrels and white rats to deal with plagues of these two animal species. Danysz (9) isolated from field mice an organism which presumably is that now known as S. enteritidis var. danysz and found it a useful means of getting rid of rats from farms, shops, and other infested places, provided he took steps to maintain its virulence, which passage through rats did not enhance but decreased. Danysz reported that in at least 50 percent of occasions on which it was used the rats were wiped out. In about 20 percent the treatment was ineffective; and, in the remainder, the results were moderately good. As young rats were more susceptible than older ones, treatments were most effective in spring and autumn. It was used not only in Paris where Danysz worked at the Pasteur Institute but also in Lille by Calmette of the Pasteur Institute there, in Hamburg by Abel, and in Copenhagen by Th. Madsen. After these early trials suspensions of the Danysz bacillus were widely used to deal with straight-forward infestations of rats, and also as a measure for the control of plague (10). In the plague epidemic in Odessa in 1902, the whole city and the surrounding villages — an area with a radius of 10 or 15 kilometres — were treated. Over a period of 3 weeks, 25,000 litres of what is described as "the virulent bouillon" were prepared in large boilers; each day's batch was distributed as soon as it was prepared. Crusts dipped in the suspension were freely distributed by the population and often left lying in the courtyards and streets. Despite this, no illness was reported either in human beings or domestic animals, but the rats completely disappeared. Danysz concluded that the experience in Odessa showed that, provided the virulence of the cultures was adequately maintained, the rats in any large city could be destroyed "without causing any danger whatsoever to its other inhabitants, human or animal, always supposing that the operations are conducted in an orderly and methodical manner" (10).

An annotation in the British Medical Journal (11) describes the paper in which Danysz reported his Odessa and other experiences as an "exceedingly modest statement of an excellent piece of bacteriological work of which the practical utility . . . . has been more widely recognized hitherto on the Continent than in England."

Later Danysz (12) described the use of his salmonella in a plague of field mice in France. In 1903 and 1904, about 600,000 litres of a suspension were prepared and distributed at a rate of about a litre per hectare. One litre of the suspension was mixed with about 1.5 kilos of crushed grain. The mixing took place on the floors of barns, schoolrooms, and inns. Men, women, and children collected the mixture in baskets and any other available container and scattered it by hand in the fields. Containers, clothes and hands became heavily contaminated. No cases occurred in human beings or in animals and birds, domestic and wild, which ate the grain freely. The mice died in large numbers. Danysz considered that in the light of these findings there was very little proof of the pathogenicity of Gaertner's bacillus and similar organisms and suggested that in food poisoning the ptomaines were the important cause.

By 1908, at least four different preparations were on sale in England; and, in that year, Dr. Collingridge, the Medical Officer of Health for the City of London, described an outbreak of illness in 12 men who had a common meal in one of five dining-rooms of a large business establishment. In this room and the adjacent pantry, a "much-advertised" virus for the destruction of rodents had been used — spread on pieces of bread. An organism from the stools of the patients was compared by Klein (13) with the organism cultured from the tube of "virus" and was said to be similar in all respects.

The makers of the four preparations stated on the labels that the bacteria were harmless to human beings and domestic animals (one said this had been shown by experiments). All the same they said the preparation "should be placed out of the reach of children and sucking calves" and they added "wash your hands after use" and "there need be no hesitation in using the fingers when preparing the bait, but they should be free from wounds or sores." Collingridge remarked that it was clear that the sellers were not sure that the preparations were harmless to children or calves or that contamination of the hands would not cause mischief. The city authorities sent the report of their Medical Officer of Health to the Local Government Board (the forerunner of the present Ministry of Health) with a strong recommendation that restriction should be put on the sale of these preparations to the public. According to custom in matters of this kind, after a due interval a question was asked in parliament. Would the President of the Local Government Board say "if his attention had been called to the report . . . . that a vermin poison advertised as harmless to human beings . . . . had caused serious illness . . . . and . . . . would he consider the desirability of placing restrictions on its sale?"

The President of the Local Government Board — also in traditional fashion — said in his reply no more than was absolutely necessary. "The administration of the Acts relating to the sale of poisons rests with the Privy Council. I will communicate with the Lord President." And no doubt this communication was duly made. And some 50 years later, by voluntary action of the manufacturers, "rodent viruses" were withdrawn from the market. In the interval, they continued to be widely used; and, when Leslie (14) examined the products most commonly used at that time, three were of the same name as those examined in 1908 by Klein and his colleagues.

Despite their widespread use over half a century there was very little conclusive evidence of human infection. Collingridge's report has been mentioned. Other reports in which there were more or less strong suspicions appeared from time to time but in only one was the evidence reasonably sound. Brown and Parker (15) reported two cases due to *S. enteritidis* var. *danysz* in persons who almost certainly ate vanilla custard cakes from a bakery in which "rat virus" had recently been used. The strain was isolated from the spleen of a mouse caught in the bakery.

THE PRESENT

These notes indicate clearly the complexity of the ecology of salmonellosis and the difficulty in successfully studying the many aspects of the problem. In the first 30 years of the present century, knowledge was derived mainly from the study of outbreaks. This gave useful information but was of limited value in determing the sources of infection and the modes of spread. Savage (16) summed up the situation in a paper he presented at the Royal Society of Medicine entitled "Unsolved Problems in Salmonella Food Poisoning." He said most food poisoning in England and Wales was due to salmonellae and that no conspicuous decline was occurring. The sources of infection were still unknown; outbreaks were rarely associated with sick animals; human carriers were seldom conclusively incriminated; case-to-case infection was not common. He thought animal carriers might be a frequent source of infection but said that there was not much evidence to support his hypothesis.

Since then knowledge of the ecology of salmonellosis has greatly increased thanks to national and international collaboration between veterinarians and medical workers. The protean nature of the salmonellae, their ubiquitous distribution, and their wide host range are now well known. They have been found in every country and isolated at one time or another from almost every warm and cold blooded living creature. Some types are host-specific, others show a variable pathogenicity for a variety of hosts. The living gut may not always be necessary for their survival. Steiniger (17-19) has produced evidence that they can exist and multiply in the partially enclosed waters of harbours in the North and Baltic Seas and in the Mediterranean. Others have isolated salmonellae from coastal waters in other parts of the world and have found these waters to be a source of contamination of shell fish nd feedingstuffs.

The foods and feedingstuffs most frequently contaminated have been identified, and effective means for rendering them safe have been or are being worked out. Much of what was not known when Savage spoke has now been elucidated, but we have to echo his words that so far there is no evidence of a conspicuous decline in incidence. Why is this? There are a number of reasons. One is that though practicable measures to control the spread of infection have been advocated for years, most of them when applied at all have been applied only recently and as yet have had little effect. Another is that the expert has been too engrossed in the intriguing problem of the exotic type and has tended to pay too little attention to the common types.

In the United Kingdom, records of the strains isolated from clinical cases have existed since 1923. Between 1923 and 1960, 179 identified types were isolated from at least one incident — an "incident" being defined as *either* a single sporadic case *or* an outbreak of any size. Of these 179 types, 104 (58 percent) were responsible for only 1.7 incidents each. The other 75 types were responsible for an average of almost 600 incidents each. But there were wide variations from the mean and 90 percent of all incidents were due to only 7 types: *S. typhi-murium* (responsible for 76 percent of incidents), *S. enteritidis*, *S. thompson*, *S. newport*, *S. heidelberg*, *S. anatum* and *S. bovis-morbificans*. Five are indigenous types; they have been isolated regularly since records were first kept. *Salmonella anatum* was first isolated in 1940 and *S. heidelberg* in 1953. These two strains, therefore, have found the British human gut in some way a more congenial place than have all but five other competitors. Another characteristic of the common types is that they are all very common in meat and eggs and their products. We do not know why the seven are so much more important than the others; but, in the United Kingdom, control of a few types would change salmonellosis from a very common to a very rare disease.

Study of the rare types should not of course be given up. Comparison of strains which become readily established with those which fail to do so or do so with difficulty might have a practical value in the control of salmonellosis and might also shed some light on the behavior of the other enterobacteriaceae and of pathogens belonging to other bacterial species. Little has been done since the work by McCullough and Eisele (20-23) on the factors which determine the occurrence of clinical disease. Their studies suggested that though small doses were sufficient to cause infection very much larger doses were required to cause illness. They observed that the number of organisms necessary to cause illness with some serotypes was 15 times greater than with others, and they also found very great differences — up to fivefold — between strains of the same serotype. Some evidence from natural infections agrees with that obtained in these volunteer studies, and some does not. The spread of infection by means other than foodstuffs in hospitals indicates that only a small dose may be necessary to cause illness as well as infection. This is often true for children and is true at least sometimes in adults. Datta and Pridie (24) investigated an outbreak due to *S. typhimurium* mainly in adults in a hospital with about 650 beds in England. It lasted over 20 weeks; 102 clinical cases occurred, and there were at least 150 symptomless excreters. How the infection was originally introduced was not determined. First one or two persons were infected; and, after an interval, cases cropped up again in several different parts of the hospital. There was a considerable amount of evidence that the infection was not food-borne. Spread probably occurred by contact, ward dust, contaminated sheets, and so on; none of them are means which permitted the rapid multiplication of the organism.

McCullough and Eisele used organisms cultured artificially, and this raises the possibility that the very large numbers required to induce clinical infection might be a consequence of the loss of some factor during culture.

The real explanation for these sometimes contradictory findings is not known, but accurate direct observations under field conditions is very necessary if we are to obtain reliable information on the dosage of organisms necessary to set up infection and disease.

THE FUTURE

These are some research projects which would increase scientific knowledge about the salmonellae. Those who work on the subject could propose other, probably more important projects. But enough is now known about the behavior of the salmonellae to reduce, within small compass, the amount of disease they cause in man and animals. Our main efforts should be directed towards ensuring the application of effective preventive measures based on what we know.

More perhaps than in most diseases, collaboration is required for the effective handling of the salmonella problem. Within countries, it is necessary to have cooperation between veterinarians; workers in preventive medicine; producers of protein feedingstuffs and of meat and its products, eggs and egg products, and other potentially contaminated foodstuffs. In one or two countries, this cooperation has been effectively developing; in some others, it is improving slowly; but, in most countries, it does not yet exist and is not likely to do so for many years to come, because veterinary and medical services are inadequate and the medical services are more than fully occupies with far more serious problems — malaria, tuberculosis, helminthiasis, smallpox, the diarrheal diseases of the young child, and, above all, malnutrition and undernutrition. Countries which import feedingstuffs and foods can protect themselves to a large extent by arranging for the routine examination of cargoes before they are distributed. Home-produced feedingstuffs and foods have to be similarly controlled.

Countries which individually are making considerable efforts in investigation and control could assist each other greatly and could reduce the volume of their work if they would set up a system of exchange of information on the prevalence of the disease in man and animals, on the sources of salmonellae, and on the serotypes they isolate. Full benefit from such an exchange depends on information being provided freely by medical men, veterinarians, and as far as possible producers, and on setting up agreed methods for the collection, recording, and analysis of the information.

In this "National" Conference there are workers from four nations. I know they already exchange information, but this is an excellent opportunity to review the methods used by each of them and to bring them nearer to a common pattern. When this pattern is established, they could invite other countries to provide similar information and to join in the exchanges. By this means, it would be possible to build up a sound scheme for the collection and dissemination on what is, to a greater extent than most diseases, an international problem.

You may observe that I have put the onus on the four countries represented here to form a nucleus round which an international exchange scheme of information on salmonellae should be built. My experience is that such a scheme, initiated by those with special knowledge and experience, is most likely to succeed and to attract experienced workers from other countries to collaborate in it.

In the World Health Organization, salmonellosis comes partly into the sphere of interest of the Unit of Veterinary Public Health and partly into that of the Unit of Bacterial Diseases. The Chiefs of these Units would be very pleased to be associated with such a scheme for the exchange of information and would do their best to help in setting it up and extending it to interested groups of workers wherever they exist.

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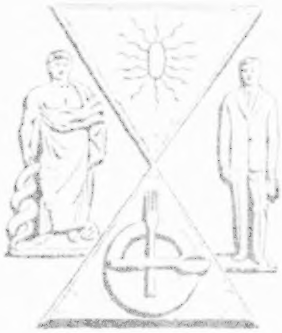

SUMMARY

Dr. Alexander D. Langmuir

Summary

Alexander D. Langmuir, M.D.[1]

In planning this conference, it was believed wise not to plan for a series of formal resolutions or recommendations but rather to rely on the proceedings themselves to serve as the most useful record of these deliberations. It was deemed worthwhile, however, that some effort be made to summarize the main problems encountered. This assignment has fallen to me. While it is probably impossible to achieve a true consensus, an attempt will be made to examine some of the broader issues that have arisen. In doing so, I may well introduce some of my personal views.

First of all, I would like to say that this conference is a good example of the philosophy we espouse here at the Communicable Disease Center in all of our general activities. We term this surveillance. This approach to public health goes back more than a hundred years. With deference to our several visitors from the United Kingdom, I would like to credit Dr. William Farr as one of the greatest practitioners of this concept. As First Secretary in the General Register Office in Great Britain, he established the system of recording vital statistics and collecting basic health facts. He published his brilliant analyses of these data in regular weekly, quarterly, annual, and special reports for the information of the general public and as the basis for the wisest administrative action.

In our various programs of surveillance here at CDC, we are guided by the basic principles established by Farr. We have faith that in a democracy an enlightened public will see that solutions are found and corrective steps are taken to all problems that are understood. It is our function in conducting national surveillance programs to collect the facts and disseminate them to all who need to know.

Repeatedly at this conference we have heard the assertion, "We know so much about salmonellosis that it is going to take a long time to control the problem." I would like to state my strong opposition to that philosophy. In 1935, we thought we knew a lot about malaria. It was a desperately serious problem. Anyone at that time who advocated the concept of malaria eradication would have been scoffed at in scientific circles. Yet, by 1945, malaria had disappeared as an indigenous disease in the United States. In 1950, poliomyelitis was a dreaded epidemic disease recurring every summer. There was no sound theoretical basis for control. Our knowledge of the immunology and pathogenesis of the disease and our capacity to grow the polioviruses had not developed to the point where any logical control actions could be undertaken. Yet, in less than 15 years, the disease has practically been eliminated. I do not accept the defeatist view that "it is going to take a long time to control salmonellosis."

I will say that we are going to have to learn more about salmonellosis if we are to be successful. For example, we do not even know whether the problem is increasing. This has frequently been argued during this meeting, but I question that the evidence is convincing. It seems entirely possible to me that the 18,000 positive salmonella cultures during the past year may have resulted from enhanced general interest and markedly improved reporting. Actually, I do not consider this an important question. We have abundant evidence of gross underreporting; the 18,000 isolations are only a fraction of the total infections which occurred in 1963. It seems likely that with the extent of the interest and the expansion of laboratory services there will be further increases in reported infections.

I do not believe that the State of Washington has uniquely less adequate sanitation than other States. On the contrary, the unique feature about Washington is the presence of a capable epidemiologist, Dr. Ernest Ager, who goes to the field and digs into problems. This same pattern can be found in any State of the Union. We can be confident that similar situations to the one in Washington will be repeated often. Whether recent events represent true increases or better recognition is not the main issue. Certainly the problem is big enough for all of us to pay serious attention to it.

There has been some discussion of the host-parasite relationship, but I have been really quite astonished at my medical colleagues, many of them leaders in the field of infectious diseases, because they do not talk with assurance about the pathogenesis of the disease in man. Likewise, I tried but failed to learn during the past 3 days about the pathogenicity of this infection in animals. What is the exact portal of entry? Is it limited to the lymphoid tissue

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of the small bowel or is the tonsil also a vulnerable tissue? Does airborne infection play a role in natural infection? What is the dose response by age? What are the factors leading to unusual susceptibility or resistance? The occurrence of cases in postgastrectomy patients suggests the importance of acid in the gastric juice, but we need to have a much more detailed understanding of these host-parasite relationships. Many basic questions remain unanswered.

Balancing this admission of ignorance regarding the pathogenesis of the infection is the amazing lack of agreement regarding the extent of the economic problem. I sense from various statements of animal husbandry and poultry experts here that there are only poor estimates of the extent to which salmonella infection causes economic losses in the industry. Here we have not a matter of human discomfort, but of dollars and cents.

This issue is crucial. If salmonellosis is a definite economic problem, then funds from many sources, both private and public, should become available to work toward solutions. It will be profitable. Leadership should come from industry. If, on the other hand, there is no substantial economic loss but only a human health problem then the primary funding and direction of effort for both research and control must come from government.

While the issue is moot, from my vantage point it is difficult to dismiss the economic aspects of salmonellosis. There would seem to be reasonable evidence from analogy to the pullorum problem, from the turkey problem, from Salmonella thompson in breeder chicks, and from the abundant episodes of contaminated food products, to conclude that sufficient economic loss occurs to warrant serious attention.

The recent hospital infection experience presents another facet of the many-sided problem; it causes acute embarrassment to the medical and health professions. It certainly piques our pride that in these days of heart surgery, artificial kidneys and organ transplants, we cannot take dominance over a miniscule little bacillus with a tail on it that gets into our hospitals, causes no end of trouble, and has us stumped. This whole situation is an indication of the extent to which fashion dictates where the money is spent and to what questions the brains of research workers are applied. In recent times, infectious diseases have been relatively ignored. The hospital salmonella problem calls for more effort. The solution may be difficult to find.

I have been asked to comment on what is the position of the Public Health Service regarding the recommendation on the use of raw eggs in hospitals. The statement of the Surgeon General on July 11, 1963, still stands, and I know of no plans to modify it. I am sure there would be utmost willingness to consider appropriate changes on the presentation of adequate evidence; but, from what has been reported here during the past 3 days, there would seem to be little basis for revision. Patients in a hospital represent a highly susceptible population. Eggnog, mixed in bulk and allowed to stand for some hours in ward kitchens and at the patient's bedside, is an ideal culture medium for bacteria. It would seem prudent to avoid using, among hospital patients, any product that may be even remotely contaminated with salmonella.

The issues of industry self-regulation or government controls have been rather fully outlined during this conference. Perhaps one or two other points might be mentioned. In public health at all levels, we have an aversion to regulations. We favor education and persuasion. Basically, the concept of surveillance is the key function for us to perform.

The Food and Drug Administration, however, is built on strong regulatory powers. I believe the Department of Agriculture has both regulatory and educational powers. One thing must be clear; all of us who serve in the Federal Government are responsive to Congress. We carry out what it legislates. Congress in turn is responsive to the public. If serious problems appear and remain unmet, Congress can be expected to investigate and legislate.

When it comes to immediate steps that need to be taken during the next year or so, I have no detailed blueprint to outline but only a few general principles to offer.

First, we must have better reporting. The Salmonella Surveillance Report represents an effort to encourage this. It has grown with the warm support of the State epidemiologists and laboratory directors. There is much additional information. Some is now being sent to us by Federal and State agriculture and food and drug laboratories, but this reporting is by no means complete. Little information has yet been received from industry. The Salmonella Surveillance Report is a special type of government document. It is not a news release, it is not an official publication of record, but it is a means of prompt communication of significant current information to those having a need to know. Anyone with a legitimate interest is welcome to receive the report and urged to make contributions to it.

Second, I would urge State health authorities to re-examine their regulations and modernize their procedures for detection and management of carriers. Much effort of field workers and laboratory services are misdirected to unprofitable routine activities. More sophisticated epidemiological approaches directed to specific problems would be rewarding.

Third, serious efforts must be made to eliminate salmonella from foods and feeds. While it may be true that the removal of this constant source of wide dissemination of small numbers of salmonella might not eliminate the whole problem, there is the real possibility that a major improvement would result. The dividends both from economic savings to industry and from reduction of human illness could well exceed expectations. No one can now say to what extent contaminated feeds may be a chief reservoir of infection from which new introductions and repeated reinfections are spread throughout the country.

Fourth, the desirability of more cooperation in international surveillance makes plain good sense. There should be a greater flow of pertinent information among the actual workers in the field, the epidemiologists, laboratory workers and key persons in industry. Furthermore, an exchange of personnel would be highly desirable. The World Health Organization is the ideal international agency for encouraging these types of undertakings.

Finally, the real achievements of this conference are twofold. The participants have gotten to know each other in a way that has rarely been possible in previous years. Each has found that respectively the other does not have horns, cloven hoofs, and a forked tail. There has been major exchange of information. All of us are better informed on the nature and extent of the problem and on the degree of activity that is under way, seeking for better definitions and solutions of the manifold problems.

The proceedings of this conference will be the record of these achievements. Let us hope that when further progress warrants, another conference can be assembled to report on progress toward our common goal. Let us hope this can come sooner rather than later and that in this interval a major reduction in the amount of salmonellosis in the country will have been achieved.

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* U. S. GOVERNMENT PRINTING OFFICE : 1965 O - 767-177

RECEIVED
NATIONAL TECHNICAL
JAN 18 1973
INFORMATION SERVICE

Public Health Service Publication No. 1262

JUN 25 1965